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Control of Vegetation on Utility and Railroad Rights-of-Way

Commonwealth of Massachusetts
Final Generic Environmental Impact Report
January 1985

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HARRISON BIOTECH
Cambridge, Massachusetts

A GENERIC ENVIRONMENTAL IMPACT REPORT
ON
THE CONTROL OF VEGETATION
ON UTILITY AND RAILROAD RIGHTS-OF-WAY
IN THE
COMMONWEALTH OF MASSACHUSETTS

by

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for

DEPARTMENT OF FOOD AND AGRICULTURE
COMMONWEALTH OF MASSACHUSETTS

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ABOUT THIS REPORT

This Generic Environmental Impact Report (GEIR) examines the practice of vegetation control on utility and railroad rights-of-way in Massachusetts. The report focuses primarily on the use of herbicides, but also describes other control alternatives such as manual and mechanical control, as well as the potential for biological control on rights-of-way.

The topics to be discussed in this report were identified by an informal survey of interested parties, including environmental groups, railroad and utility companies, local and state officials, and others. These individuals and groups were asked to identify issues which they felt should be addressed in the study. This process resulted in a broad range of topics which have been covered in this Generic Environmental Impact Report. Since the report had to be completed in four months, however, not all the topics could be addressed in equal detail. Most of the available resources were allocated to the development of a scientific base of information regarding the toxicity and mobility of fourteen herbicides used in Massachusetts for vegetation control on rights-of-way.

The report is divided into three sections:

1. The main body presents an overall discussion of the major topics of this report such as alternative control measures and descriptions of vegetation control problems.
2. Appendix I presents supplemental information on a variety of topics.
3. Appendix II presents a literature review on the fourteen herbicides. For each herbicide in Appendix II, the following information is presented:
   - Acute and subacute/subchronic toxicity by oral, dermal, and intraperitoneal administration (where data are available), as well as information on eye and skin irritation;
   - Special toxicological studies, including carcinogenicity, teratogenicity, and mutagenicity;

- Fate in soil and water, including leaching potential, dissipation times, run-off potential, and degradation rates; and
- Effects on non-target organisms, including birds, fish, lower aquatic organisms, bees, and other organisms.

Summary statements about the literature are included in the main body of the report. These summaries are purposely brief so that the reader will not be tempted to rely on them, but rather on the full complement of information in Appendix II. Accompanying the summary statements in the main body of the report are general discussions which interpret the data presented in Appendix II. These discussions also present site-specific factors that must be incorporated into an analysis of impact.

Secondary source material (i.e., reviews of original studies) was used whenever possible because of the limited time available for the preparation of this report. Secondary source material was found to be adequate in assessing the acute toxicity of the herbicides, as well as their impact on non-target organisms. However, this material was found to be uneven in its coverage of important topics concerning the mobility and persistence of the herbicides; primary sources (original studies) were obtained to supplement where necessary. In regard to chronic toxicity, the secondary source material was considered inadequate and primary sources were used in most instances. Primary and secondary sources are identified in the bibliography at the end of this report.

OVERVIEW

Public concern is growing about the introduction of chemicals into our environment. Lately, herbicides have been given particular attention because of the controversy regarding the use of Agent Orange in Vietnam. (Agent Orange, named for the color of the storage drums, is a 50:50 mix of butyl esters of 2,4-D and 2,4,5-T and contains a type of dioxin that is known to cause serious health effects.) In Massachusetts, railroad and utility rights-of-way have become the focus of concern about herbicides despite the fact that herbicides are used for many other purposes. Before presenting information regarding vegetation control on rights-of-way, it is important to understand the overall picture of herbicides usage in Massachusetts and the significance of their use on rights-of-way.

In Massachusetts, herbicides are used in agriculture, on residential lawns and gardens, on parks and recreational land, golf courses, and on commercial grounds. For each of these uses, Table 1 shows the estimated acreage treated with herbicides, the total amounts of herbicides used (as pounds of active ingredient), as well as major herbicides in each market. This information was generated from interviews with distributors, large scale sellers of herbicides, lawn service firms, members of the Cooperative Extension Service, and other members of the technical support community. Because of the lack of a reliable data base regarding the quantities of herbicides used in Massachusetts, most estimates are presented as ranges of values.

Agriculture represents the biggest use of herbicides in Massachusetts, with an estimated 161,000 to 320,000 lbs of herbicide used on 193,000 acres (rate of usage = 0.83 to 1.66 lbs/acre). Within agriculture, the production of field corn and cranberries uses the greatest amount of herbicide. Another major use of herbicides is on residential lawns, where 55,000 to 110,000 lbs of herbicides, usually "home and garden" formulations, are applied to a total of 160,000 to 210,000 acres (rate of usage = 0.26 to 0.69 lbs/acre). Lastly, herbicides are used on

TABLE 1

ESTIMATED HERBICIDE USAGE IN MASSACHUSETTS,
SELECTED USE CATEGORIES

Use Category	Estimated Acreage	Herbicide Usage (lbs. A.I.)	Leading Herbicide Products
Agriculture	193,000	161,000-320,000	
Field Corn	45,000	90,000-160,000	Atrazine, alachlor, metolachlor
Sweet Corn	7,500	14,000-30,000	Atrazine, alachlor, metolachlor
Potatoes	3,500	7,000-20,000	EDTC, endothal
Cranberries	12,000	40,000-80,000	Dichlobenil, napropmamide
Other (Hay, tobacco, minor vegetables)	125,000	10,000-30,000	Numerous
Residential Lawns*	160,000-210,000	55,000-110,000	2,4-D benefin (minor)
Minicipal and Institutional Landholdings	100,000-127,000	36,000-70,000	
Parks & Recreation**	42,000	6,000-10,000	Dicamba, 2,4-D, benefin, MCPP, DCPA
Golf Courses	28,000-35,000	18,000-30,000	Dicamba, 2,4-D, benefin, MCPP, DCPA
Commercial	30,000-50,000	12,000-30,000	Dicamba, 2,4-D, benefin, MCPP, DCPA
Right-of-Way Use	14,729	100,600	
Total Selected Uses		352,600-600,600	

*Does not include garden use which is very small. **Does not include forested land or water.

municipal and institutional land holdings, primarily on golf courses and commercial grounds. A total of 36,000 to 70,000 lbs of herbicides are applied in this category to an estimated area of 100,000-127,000 acres (rate of usage = 0.28 to 0.70 lbs/acre).

The use of herbicides on rights-of-way contributes roughly 17%-29% of the total use of herbicides in Massachusetts, with an estimated 100,600 lbs applied in 1981 to 14,729 acres (rate of usage = 6.83 lbs/acre). Within the rights-of-way category, the contributions from railroad, utility, and highway rights-of-way are shown in Table 2. Railroads use the most herbicides in terms of pounds of active ingredient (49,100 lbs used by railroads in 1981 as compared to 35,000 lbs and 32,400 lbs for utilities and highways, respectively. The rate of use is highest on railroad yard and line maintenance (12.8 lbs/acre) and lowest on railroad brushwork (3.5 lbs/acre).

All data, except that provided by one utility company, are for 1981. That year was considered to be more representative than recent years because of the supension of treatment in certain areas pending resolution of regulatory questions (see Appendix I, Chapter I).

This report focuses on railroad and utility rights-of-way, since the use of herbicides on highways has decreased considerably in the last few years due to budget constraints. Information from the Massachusetts Public Works Department indicates that herbicide use (in pounds of active ingredient) decreased by about 40% from 1981 to 1982. At the same time, the percentage of roadway treated with herbicide decreased from about 4.2% to about 1.8%. (The rate of usage, however, increased from 5.96 to 8.39 lbs/acre.) The purpose of vegetation control on highways is to aid in snow removal, reduce snow drift, reduce maintenance costs in ditches and shoulders (including the need to keep guardrails clear), control poison ivy, and increase the safety of motorists. Table 3 shows the herbicides used for state highway maintenance in 1981 and 1982. In both of those years, about 6400 acres were maintained by mowing. Additional information about the vegetation control on highways is provided later in this report (see Information Requested by Reviewers).

TABLE 2

COMPARISON OF HERBICIDE USAGE BY TYPE OF RIGHT OF WAY (1981 DATA)

Type of Right-of-Way	Pounds of Herbicides	Acres Treated	lbs/acre	Contribution to total herbicide use in Mass.
Railroad	49,100	3845*	12.76	8-14%
			(yard, branch, and main line)	
			3.5 (brushwork)	
Utility	35,000	8120	4.3	6-10%
Highway	16,500	2764	5.96	3-5%
Total	100,600	14,729	6.83	

* Includes 120 acres of Amtrak line

TABLE 3

HERBICIDES USED ON MASSACHUSETTS DEPARTMENT OF PUBLIC WORKS HIGHWAYS

	1981		1982	
Herbicide	Amount	Acreage	Amount	Acreage
Karmex® (diuron)	5425 lb	207	4694 lb	180
Aminotriazole	6905 lb	1584	1717 lb	394
Fenavar® (aminotriazole, bromacil, and fenac)	286 lb	384	231 lb	310
Dowpon M® (dalapon)	3207 lb	127	2789 lb	110
Krenite®	399 lb	388	103 lb	100
Weedone 170® (2,4-D and 2,4-DP)	118 lb	41	147 lb	51
Spike® (tebuthiuron)	95 lb	25	10 lb	3
Fenavar®	41 lb	8	80 lb	16
Total acres		2764		1164

Total Acreage = 65,000 (approximate).

Historical Practices on Railroad and Utility Rights-of-Way

The most significant event in the history of vegetation control on rights-of-way has been the development of herbicides. Although herbicides had been around since 1850, they did not become commonly used until the mid-1940's when 2,4-D was discovered, initially as a plant growth stimulant and then as an herbicide. In 1948, another phenoxy compound, 2,4,5-T, was introduced and immediately found extensive use along rights-of-way because it was more effective against

woody plants than 2,4-D. After these two chemicals came onto the market, a large number of additional herbicides were developed. By 1950 there were 15 herbicide active ingredients on the market. By 1980 there were 180 herbicide active ingredients available in 6000 formulations.

On railroad rights-of-way, a variety of methods were used before the introduction of herbicides in the 1940's. These included

- application of waste oil to the ballast area
- heavy applications of salt
- controlled burns 3 to 4 times a year
- applications of arsenical compounds

After 1950, however, herbicides quickly became the primary means of control in railroad yards, and on branch and main lines. In Massachusetts, brush control along the sides of the railroad continued to be done manually until the early 1970's, when herbicides became the preferred alternative.

On utility rights-of-way, vegetation was controlled manually before the 1940's. Undesirable vegetation was cut with axes and brush-axes on an average of every three years. The workers who performed the cutting came from Quebec, Canada, and lived on the rights-of-way in tents or trailers during the cutting season. Chainsaws and brush-saws began to be used on the rights-of-way in the late 1940's. At the same time that herbicides were introduced, the availability of the Canadian woodcutters decreased as the economy of Quebec improved and immigration laws became stricter. The conversion from cutting to herbicide control began around 1955, at which time spraying was done mostly by hydraulic sprayers mounted on four-wheel-drive trucks or all-terrain tractors. Helicopters were used in some areas of difficult terrain in the western part of Massachusetts. In the early 1960's selective basal and foliar treatments (explained below) began to be the primary treatment methods. Helicopters were last utilized in 1971.

Vegetation Control Problems on Rights-of-Way

Utility and railroad rights-of-way present considerably different vegetation control problems, and different approaches are used to solve those problems. On utility rights-of-way, only a subset of the vegetation needs to be removed, namely trees and woody growth around structures. Trees must be removed because they can fall into lines or cause "flashovers" or "arcing" between the trees and the line, causing breakage or short circuits. The allowable potential heights of trees varies according to the heights of conductors, allowing for sag under ice-laden conditions. Allowable heights are 10 feet for 345 kV lines, 8 feet for 115 kV lines, and 20 feet for side strips (where tree falls could cause a problem). Vegetation control on utility rights-of-way is therefore selective, attempting to eliminate trees whose potential height is above acceptable levels, and preserving vegetation whose potential height is below this level. Around structures and on access roads, target vegetation includes all woody vegetation that may hinder routine inspection and maintenance of the line. In these areas, non-target growth is limited to herbaceous species.

A different situation exists on railroad rights-of-way where the goal is to eliminate all vegetation in train yards and on branch lines and main lines. If allowed to become established on the right-of-way, vegetation could increase the amount of organic matter under and around the track, resulting in water retention, drainage problems, and an increased rate of decay of wooden ties. The requirement to maintain a vegetation-free area, or even to severely restrict vegetation, demands a different approach to vegetation control than that practiced on utility rights-of-way. There is no possibility of selectively treating desirable and undesirable vegetation. A number of different active ingredients are generally used in combination so that the maximum number of target species can be eliminated. (On utility rights-of-way, applications involve usually only one, sometimes two, active ingredients). The mixtures of different herbicides are applied evenly over the lines and yards, rather than applied on certain plants.

"Brushwork" on railroad rights-of-way, on the other hand, is similar in many ways to the control of vegetation on utility rights-of-way. Brushwork involves the control of woody vegetation adjacent to the ballast. The width of the brush control area depends on the location and height of potentially dangerous trees, but is commonly 20 to 25 feet. The purpose of this activity is to maintain visibility and prevent the disruption of communication lines. Brushwork involves the selective treatment of tall-growing and the encouragement of desirable low-growing shrubs and herbaceous growth.

THE HERBICIDE ALTERNATIVE

The following section discusses the use of herbicides on railroad and utility rights-of-way. It describes current practices, and then introduces the individual chemicals that are commonly used. Most of this section is devoted to a discussion of the toxicity and mobility of these herbicides and ways to minimize their impact.

CURRENT PRACTICES

Railroad Rights-of-Way

Herbicides are applied to lines and yards by means of a high-rail vehicle which can move both on and off the track. The vehicle moves 10 to 15 miles per hour and sprays herbicides from lateral arms located 12-18 inches off the ground. The nozzles are spaced 20-24 inches apart and have typical flow rates of 2-3 gallons/minute at 40 pounds per square inch pressure. Both pre-emergent and post-emergent herbicides are used. Pre-emergent herbicides, such as metolachlor, are ones that are applied before the weeds emerge. On rights-of-way in Massachusetts they are applied in April and early May. Post-emergent herbicides, such as 2,4-D, are ones that are applied after the weeds emerge. On rights-of-way in Massachusetts post-emergent herbicides are applied in June and July. Different types of lines (e.g., main lines vs. branch lines, branch lines with and without ballast, etc.) receive different treatments, either pre- or post- emergent, or both. Since it prevents any vegetation from emerging, pre-emergent treatment is used where control of vegetation is particularly important--in yards where slippage would endanger yard workers, for example.

Typical herbicides used on railroad yards are Atratol® (atrazine), and Karmex® (diuron). Branch lines may be treated with these herbicides or with 2,4-D, diquat, ametryn, or Banvel 720® (a combination of dicamba and 2,4-D). Usually three or four of these products are mixed together and applied with a water solvent. The total amount of applied

material is on the order of 40 gallons/acre; the number of pounds of active ingredient per acre varies with the particular combination of products used. (A double track 35 feet wide equals roughly 4.25 acres/mile). Applications in yards and on branch and main lines are made every year.

In brushwork, the high-rail vehicle is equipped with nozzles (at the end of lateral arms) designed to limit the drift of the material to the edge of the right-of-way. In Massachusetts, a typical mixture used in brush control is Roundup® (glyphosate) combined with Garlon 3A® (triclopyr) mixed with water. Twenty-five gallons of mixed material is used per acre.

Utility Rights-of-Way

Herbicides are applied to utility rights-of-way by a variety of application techniques:

Basal spraying is the application of herbicide, usually in an oil carrier, to the root collar, exposed roots, and the lower 18 inches of the trunk. The material is released as a directed spray of a large droplet size. Basal spraying can be done year round (except in deep snow) by backpack or hydraulic sprayer.

Foliar spraying is the application of herbicide, usually in a water carrier, to the leaf surfaces of the entire plant. In order to assure maximum coverage the material is often released in a "mist" of small droplet size. Larger droplet sizes are also effective, however, and thickeners that increase the droplet size are often added. Surfactants may also be added since they increase the spread of the material on the leaf surface. Foliar applications are limited to summer months, along with late spring and early fall, and can be done with a backpack or hydraulic unit.

Cut stump treatment is the application of the herbicide to the cut surface of a stump, and sometimes the root collar and exposed roots. In Massachusetts, a sponge applicator has been developed which

effectively controls the amount of herbicide released. Stump treatments are not feasible when there is deep snow cover or when there is significant sap flow from the stump in late winter/early spring.

Dormant stem spraying is the application of the herbicide to all exposed wood when the foliage is absent from the plant. The root collar and all stems are thoroughly drenched. An oil carrier is used to increase bark penetration.

Dry herbicide application involves the application of herbicide in the form of pellets, granules, or beads to the soil surface near undesirable species. Rainfall moves the herbicide through the soil where it can be taken up by plant roots.

Other techniques not commonly used in Massachusetts include frilling, in which dry herbicides are placed in shallow V-shaped cuts in the bark, and tree injection, in which herbicide material is injected into the cambium layer with a device that wounds the tree and inserts the herbicide in one operation.

The herbicides used on utility rights-of-way differ considerably among utilities. In 1981, for instance, New England Power Company and Massachusetts Electric Company used primarily Krenite®, as well as mixtures of picloram and 2,4-D (Tordon 101®, Tordon RTU®), along with a mixture of Tordon 101® and Garlon 3A® (triclopyr). Northeast Utilities, on the other hand, used Ammate XNI® (ammonium sulfamate), in water, Krenite®(fosamine ammonium) in water, Garlon 4® (triclopyr) in kerosene, and Banvel CST® (dicamba) without dilution as a stump treatment.

HERBICIDES COVERED IN THIS REPORT

After consultation with applicators, utilities, and railroad companies, fourteen herbicides were found to be important in the control of vegetation on rights-of-way in Massachusetts. The following information briefly introduces these herbicides.

Aminotriazole is a post-emergent, non-selective herbicide mixed with water that slowly inhibits chlorophyll formation over two to three weeks.

Ammate® is a post-emergent herbicide mixed with water that kills plants on contact or after translocation within the plant.

Atrazine is a selective, pre-emergent and early post-emergent herbicide mixed with water that inhibits photosynthesis.

Bromacil is a pre- and post-emergent herbicide mixed with water that inhibits photosynthesis.

2,4-D is a post-emergent, selective herbicide usually mixed with water, although oil-soluble formulations and granules are sold. This herbicide kills plants by causing them to grow too quickly.

Dicamba is a pre- and post-emergent, selective herbicide mixed with water or used as granules. It kills plants as they germinate by interfering with protein synthesis.

Diquat is a post-emergent, selective herbicide mixed with water that inhibits photosynthesis and also acts as a plant dessicant.

Duiron is a pre- and post-emergent non-selective herbicide mixed with water that inhibits photosynthesis.

Glyphosate is a post-emergent, broad-spectrum herbicide mixed in water that blocks cell metabolism by inhibiting synthesis of aromatic amino acids.

Krenite® is a post-emergent growth regulator mixed with water which inhibits the normal development of leaf buds.

Metolachlor is a pre-emergent selective herbicide mixed with water that inhibits the growth of seedlings.

Picloram is a post-emergent, selective herbicide that can be used undiluted, as pellets, or, when combined with 2,4-D as Tordon 101®, mixed with water. The herbicide disrupts the formation of a number of plant tissues.

Tebuthiuron is a pre-emergent, non-selective herbicide, applied dry or as pellets, which must later be washed into the soil by rain and taken up by the roots.

Triclopyr is a post-emergent, selective herbicide mixed with water that disrupts the formation of a number of plant tissues.

EFFECTS OF HERBICIDES ON PUBLIC HEALTH

The potential hazard to humans from the use of herbicides on rights-of-way is a function of (1) the amount of harm the herbicide causes in the body (toxicity), and (2) the amount of herbicide that reaches the body (exposure). The first part of this section describes the potential toxic effects of the herbicides and the types of toxicity tests discussed for each herbicide in Appendix II. The second section is a general discussion of the potential routes by which humans may be exposed to herbicides used on rights-of-way. Finally, this section summarizes the information presented in Appendix II regarding the toxicity of the individual herbicides.

Toxicity

The term toxicity refers to any deleterious effect produced by a chemical or physical agent on a biological system. Toxicological data can be divided into four general categories: acute, subchronic, chronic, and special studies. Since special studies are often the most critical and controversial, this discussion begins with an overview of carcinogenicity, mutagenicity, and teratogenicity.

Carcinogenicity Cancer is characterized by the unrestrained growth of daughter cells from an original target cell. This target cell is assumed

to have been modified by one or more events in which the DNA and/or other cellular regulatory mechanisms were altered. Details of the cellular and biochemical events leading to tumor formation and progression to malignancy are not clearly understood. The growth and spread of cancer has been shown to depend in part on host factors such as hormonal and immunological status and genetic background, as well as a variety of modifying factors such as lifestyle (diet, tobacco use, alcohol, stress).

Three major types of information can be used to identify agents that may pose a carcinogenic hazard to humans. They are:

- epidemiologic evidence derived from studies of exposed human populations;
- experimental data from long-term tests in animals, and
- supportive evidence derived from short-term tests whose results correlate well with in vivo carcinogenic activity.

Human data provide the most secure basis for evaluating the carcinogenic effects of an agent. However, in regard to the herbicides covered in this report, epidemiologic studies are limited by imprecise data on exposure. A major source of this imprecision is the long and variable latency period (ranging for 2 to 40 years) between initial human exposure and clinical manifestations of cancer. Another drawback is the necessarily limited size and availability of test populations.

In the absence of suitable human data, animal tests (with their associated extrapolation uncertainties) are utilized to provide the best information available to assess carcinogenic risk to humans. Typically, both sexes of a species (usually a rat or a mouse) are used; the test substance is administered continually by the selected route of administration from weaning through the major portion of the animal's life or until death. The amounts of material administered are often the maximally tolerated dose and half that amount.

Evidence of carcinogenic activity for the herbicide or other test substance can be demonstrated in one of three ways:

1. By induction of a tumor type not usually observed in the test species;
2. By induction of an increased number of a tumor type normally seen; or
3. By the appearance of tumors at a time earlier than would otherwise be expected.

The uncertainties in employing animal tests to determine the carcinogenic hazard to humans are numerous. Some of the more important factors are species differences, genetic variability, metabolic capabilities, body weight, lifespan, and DNA repair capabilities. Animal studies are also often performed at high doses to ensure that a statistically significant incidence of tumors is produced in the relatively small population of test animals usually used in such tests. These results must be extrapolated to much lower exposure levels typical of human situations.

Short-term tests, which measure the induction of neoplastic cell transformation in cultured mammalian cells, can provide useful supportive information. (Neoplastic growth is new tissue growth that serves no physiologic function.) These tests are rapid, less costly, and require significantly less sample than whole animal testing. On the other hand, they are imprecise because they do not completely mimic whole body reactions. Transformation assays involve the treatment of cultured mammalian cells with materials to see if they convert the cells to a pattern of unrestricted growth. Results accumulated to date show a good correlation between transformation response in cell culture and carginogenicity in whole animal studies. The major disadvantage of these tests is the lack of reliable metabolic activation systems. This factor is important because many known carcinogens exert their influence in humans and experimental animals after metabolic conversion in the body to the active form.

Mutagenicity A mutation can be defined as any heritable change in the genetic material of a cell or organism. The health consequences of

deleterious mutation in human populations is poorly understood. It is commonly believed, however, that mutations are invariably harmful to human health. Among the possible sequelae of a mutation are cell death, altered structure and/or function, or no overt immediate effect (should the mutation be unexpressed by virtue of its recessive nature). An agent constitutes a genetic risk to future generations only if its mutagenic potential is realized in germ cells (i.e., eggs or sperm). Somatic (non-germ cell) effects are also important in that they may lead to cancer, terata, or aging phenomena in an individual, but the risk to society is less since that effect is not transmitted to future generations.

The current consensus among geneticists is that four test systems provide the highest degree of confidence in assessing mutagenicity. These are the mouse heritable translocation test, the mouse specific-locus test, the rodent dominant-lethal test, and the mouse in vivo somatic mutation test or spot test. Many of the herbicides covered in this report have been examined in these test systems. Positive results in these tests are reliable indicators that the mutagen has reached the germ cells and affected their genetic constitution in a manner that can be detected in resulting progeny.

Positive findings in one or more of the remaining battery of non-heritable genetic tests may be indicative of the possibility of heritable effects, but they do not constitute definitive evidence that a substance poses a hazard to humans. For instance, the significance of positive findings in in vitro cytogenetic tests is questionable due to the lack of repair mechanisms, metabolic processes, etc. that would be present in a whole animal system. Results in bacterial systems are even further removed from the human exposure scenario. Differences in the organization of DNA in prokaryotic (bacteria and blue-green algae) and eukaryotic (animals) organisms make it necessary to test for mutagenic capability in both systems.

Table 4 contains a list of mutagenicity tests currently in use. They are in rough order of decreasing value with respect to predicting results for germ cell mutagenesis in vivo. The list is by no means all inclusive, but includes most of the tests reviewed in Appendix II.

TABLE 4

MUTAGENICITY TESTS (IN ORDER OF DECREASING VALUE)

Test System	Comments
Mouse Heritable Translocation	Directly measures inherited chromosomal damage in a mammal. Detects reciprocal translocations (the shift of a portion of a chromosome to another part of the same chromosome or to another chromosome). These reciprocal translocations are of concern because offspring of carriers have a high probability of inheriting aneuploid genomes that can result in death and/or major defects. (aneuploid = an organism whose somatic nuclei do not contain an exact multiple of the number of chromosomes). Mutant F_1 animals are detected by deviations from normal fertility or by cytologically detected chromosome aberrations.
Mouse Specific-Locus	The only established test that directly measures inherited point mutations (affecting only one or a few DNA base pairs in a gene) in a mammal. Mutant F_1 animals are generally scored on the basis of phenotype (i.e., a detectable expression of a mutation).
Mouse Dominant Lethal	Scores all genetic events that cause the deaths of offspring as early or mid-term embryos. Since mutants are dead they cannot be genetically tested, but this test is a signal that other types of chromosomal damage are also being induced.
Mouse In Vivo Somatic Mutation (spot test)	Detects expression of recessive coat-color genes for melanocyte precursor cell of a midgestation embryo. The test is significant

	because it is an in vivo mammalian test capable of detecting both gene mutations and various kinds of chromosomal damage and because it provides evidence that heritable genetic alterations may be induced.
In Vivo Mammalian Cytogenetic	Measures chromosomal aberrations (structural or numerical). The majority of spontaneous abortions in humans are associated with chromosomal aberrations.
Sister-Chromatid Exchange	A reciprocal exchange of segments between sister chromatids of a chromosome. Significance to humans is not known.
In Vitro Mammalian Cytogenetic	Measures chromosomal aberrations induced in culture.
Drosophila Sex-linked Recessive Lethal	Detects mutations on X chromosome (∿20% of entire genome).
Yeast Mitotic Recombination	Detects genetic damage brought about by agents interfering with the function of the spindle-fibre apparatus. The target molecules are specific proteins rather than DNA.
Bacterial Reverse Mutation, and DNA Repair	These tests such as the bacterial Ames/Salmonella test and the E. coli reversion test, detect various change in prokaryotic chromosomal material.

Teratogenicity Another category of tests which are critical to the assessment of hazard is teratology. Teratology is broadly defined as the study of malformations of the newborn that occur as a result of an adverse effect on the developing fetus. The detailed biological mechanisms of teratogenesis are not well understood. Such factors as nutritional status, age of the mother, placental variations, metabolic differences, dose, and route and time of gestation at which a fetus is exposed may all influence the potential teratogenicity of a chemical in a particular species.

A number of terms are used to describe adverse effects on the developing conceptus. "Embryotoxicity" can be defined as toxic effects on an embryo during differentiation and organogenesis. "Teratogenicity" is one type of embryotoxic effect that occurs during the formation of major organs and physical structures, and results in a malformation of one or more organs or structures. "Fetotoxicity" is an adverse effect that occurs after major organs and structures are formed, and results in a toxic or degenerative effect on those organs or structures. Additionally, the severity of effect is generally considered to be in the decreasing order of embryotoxic, teratogenic, and fetotoxic effects. Fetotoxic effects (e.g., slow growth and low birthweight) are often reversible. Confusion arises when minor effects on organs or physical structures (minor teratogenic effects) are considered to be fetotoxic because they do not affect the survival of the organism.

Tests for teratogenicity generally involve the administration of the chemical to pregnant rats or mice during the critical days for teratogenic effect (days 6 to 15 in rats). Test protocols should (but sometimes do not) include a histological examination as well as an observation of visible, easily measured signs of viability.

Egg injection studies have been used to assess the teratogenicity of a number of herbicides discussed in Appendix II. In these tests, the chemical is injected directly into chick eggs, and the effects are noted on the percentage of eggs that hatch. These tests, however, have

limited relevance to human teratogenic effects because of the absence of anatomical and physiological maternal-fetal relationship.

Other Toxicity Studies Aside from the three special study areas discussed above, two other types of toxicity studies are also vital to the assessment of hazard; namely, chronic and acute toxicity tests. Chronic toxicity studies generally involve the administration of a compound for a substantial portion of the lifetime of the test animal. Such studies are designed to detect the lowest concentration that causes no apparent effect. They also detect effects on survival, growth, functional integrity of body organs, and reproductive capacity.

The next level of toxicological study is a subchronic or subacute test, which involves administration of the test chemical on multiple occasions. Experiments are generally conducted for 90 days with rats and mice, and for six months to one year with dogs. These short-term subchronic studies are typically conducted at higher exposure concentrations than chronic studies. Pathological changes may thus be more clear-cut because they occur more quickly with the higher doses and because they are not obscured by other chronic changes, such as aging.

Acute toxicity studies provide information on the effect(s) of a single exposure. Acute toxicity is generally measured by the median lethal dose (LD_{50}) or median lethal concentration (LC_{50}); i.e., the dose or concentration that will kill 50 percent of the test population under stated conditions. Lethality provides a standard of comparison among many substances whose mechanism and sites of action may be markedly different. The LC_{50} value has general acceptance as an early warning about potential adverse effects, but it is only roughly indicative, if at all, of the effects of chronic exposure to small amounts of a chemical.

Acute toxicity studies also include tests to determine local effects of chemicals when applied directly, e.g., to the skin and eyes. The major types of local effects than can occur are irritation, corrosion, and sensitization. An irritant effect is a reversible effect, while corrosion

causes visible destruction and irreversible alteration in the tissue at the site of contact. Sensitization involves an immunologic mechanism.

Exposure

There are three primary ways in which the human body can be exposed to chemicals: ingestion, dermal absorption, and inhalation. Tables 5 ranks the relative importance of these three routes to groups of people on, adjacent to, or at a distance from rights-of-way. Since no quantitative information was found on the exposure levels of herbicides to any of these groups of people, the entries in this table are qualitative judgments based on likely pathways of movement of the herbicides.

Ingestion As shown in Table 5, ingestion of herbicides can occur in a number of ways:

- Ingestion of contaminated water from nearby wells or surface waters used for drinking water (discussed in the next section).
- Ingestion of residues on food grown adjacent to or on rights-of-way.
- Ingestion of berries or mushrooms along the right-of-way.

Residues on berries may be a significant route of exposure, depending on the stage of development of the fruit and the length of time after spraying that the berries are consumed. Frank et al. (1983) measured the residues of 2,4-D on raspberries, blueberries, and strawberries, after a broadcast application of 0.8-6.0 kg/ha of 2,4-D on rights-of-way and other sites. On red raspberries, residues were negligible if spraying occurred during the flowering season. When 2,4-D was sprayed on the immature raspberries, residues of 0.2 ppm were ultimately found when the berries were ripe. When ripe fruit was treated, residues of 2.6 to 31 ppm were found immediately after treatment. Over a 2- to 5-week period these levels dropped to 0.1 and 3.3 ppm.

TABLE 5

POTENTIAL EXPOSURE TO HERBICIDES FROM RIGHTS-OF-WAY APPLICATIONS

Group	Route		
	Ingestion	Inhalation	Dermal
On the right-of-way			
Applicators	* (after dermal contact)	***	***
Others			
Berry or mushroom pickers	**	* (from volatiles)	**
Hikers; others moving through right-of-way	* (after dermal contact)	* (from volatiles)	**
Adjacent to the right-of-way			
Residents	* (from gardens)	** (very low concentrations)	*
Other activities (e.g., playground)	* (from garden berries)	** (very low concentrations)	*
Far from right-of-way			
Connected hydrologically via public water supply	* or ** (depends on herbicide)	o	* or ** (from bathing)

TABLE 5 (CONTINUED)

POTENTIAL EXPOSURE TO HERBICIDES FROM RIGHTS-OF-WAY APPLICATIONS

Group	Route		
	Ingestion	Inhalation	Dermal
Connected hydrologically but not via public water supply	o	o	* or ** (from swimming)
Not connected hydrologically	o	o	o

*** = probable under most conditions
** = probable under certain conditions
* = possible (but not likely)
o = conceivable, but highly unlikely to result in detectable exposure

On blueberries, residues ranged between 0.84 and 10.7 ppm, independent of ripeness of fruit or length of time after spraying (up to 37 days). On ripe strawberries, residues were 10.1 and 5.49 ppm in two different sites, respectively. On the strawberries with residues of 5.49 ppm, a rapid decline in residues was observed over a 4-day period (to 0.03 ppm). On the strawberries with residues of 10.1 ppm, residues declined slowly to 6.9 ppm. Picloram residues were also measured in strawberries. Ater 4 days, residues decreased from 0.23 ppm to non-detectable levels.

No data were found on the residues of herbicides that may be expected on mushrooms in rights-of-way. It is possible that residues absorbed by fungal filaments in the soil could be translocated to mushrooms. Alternatively, herbicides may be absorbed by the small primordium when it expands after a rainfall to form the recognizable mushroom. The literature reviewed in Appendix II indicates picloram and atrazine accumulate in fungi. However, no information was found on particular species or residue concentrations.

As an interesting note, one mushroom (Lepomis lentinus) is commonly found along railroad rights-of-way and is called the "trainwrecker mushroom," having a reputation for causing derailments. This is an edible but tough, woody mushroom that requires extensive cooking to soften its tissue (Miller, 1972), a practice that is also likely to reduce any herbicide residues.

Dermal contact with the herbicide by applicators may be the most significant route of exposure. Unfortunately, no estimates are available. To determine the amount of exposure, estimates must be made of the amount that reaches the skin and the amount that is absorbed through the skin. The amount that reaches the skin depends on

1. The amounts in the air, on leaf surfaces, on application equipment, and on other surfaces to which the applicator comes to contact;

2. The amount of contact the applicator has with unmixed material during pouring, mixing, opening bags, etc;
3. The applicator's clothing, i.e., the degree of protection it affords; and
4. The extent of the body, and which parts of the body,come into contact with the herbicide.

Skin uptake or absorption through the skin is estimated by knowing:

1. The duration and frequency of contact with the herbicide;
2. the area, location, and integrity of the skin exposed; and
3. the physical and chemical properties of the particular herbicide.

In regard to this last factor, the most important information appears to be the rate of diffusion of the herbicide through the stratum corneum layer of the skin. Such rates are not known for most chemicals, but estimates can be made by knowing the permeability coefficient of the skin and the partition coefficient of the herbicide between skin and water (often the octanol/water partition coefficient is used, but this method has yet to be validated).

Dermal contact can also be a route of exposure for other persons on the right-of-way. Hikers, berry pickers, birders, and others will have varying amounts of dermal exposure depending on the above factors, as well as on the length of time since the last application. Weather factors, particularly rainfall, will also be important, as well as the rates of photodegradation and volatilization of the individual herbicides.

Inhalation Inhalation of herbicide droplets and vaporized molecules is likely to be a route of exposure for applicators and, possibly, to those adjacent to rights-of-way. One of the primary factors in the determining the significance of inhalation exposure is the size of the herbicide droplet and the amount of the herbicide that is in vapor form (volitilized). Droplet size is determined primarily by the size and the shape of the nozzle orifice as well as by the pressure under which the herbicide is released. Larger droplets (>500 microns) are generally considered to be "drift safe," since they are too heavy to move through the air. This droplet size, however, can be too large for some

herbicides to be effective. Thickeners, particulating agents, and adjuvants decrease the number of fine droplets in a spray. Small droplet sizes (10 microns and less) are particularly important because of the ability of these particles to move through the respiratory system. Droplets of 5-10 microns are generally deposited in the nasal passage (where high velocities of air and changes in direction cause them to be deposited). Droplets of 1-5 micron size tend to settle out in the tracheal bronchial region. Droplets of less than 1 micron, as well as volatilized molecules, are distributed throughout the alveolar region which represents a large and highly absorptive surface.

Herbicides can also volatilize from spray droplets or from treated soil and vegetation, and move through the air in vapor form. Although some volatile forms can vaporize rapidly at 65°F, most herbicide formulations used on Massachusetts rights-of-way are low-volatile forms and vaporize at temperatures of about 80°F or above. Inhalation of those in drift, therefore, may be greater when application occurs during summer periods. The volatility of a herbicide is dependent on its surface tension, viscosity, specific gravity, and vapor pressure (Arthur D. Little, Inc., 1979). The vapor pressures of the 14 herbicides are prescribed in Appendix II.

Summary of Toxicological Literature

The following information briefly summarizes the results of the literature review presented in Appendix II.

Aminotriazole has low acute toxicity, but causes some effects (altered weight gain, enlarged thyroid glands) in subchronic tests. Aminotriazole appears to be a carcinogen, causing tumors in both rats and mice. It has been used as a positive control in carcinogenicity tests. Most available studies show no mutagenic activity of aminotriazole, although more study is needed. Insufficient data are available to assess its potential teratogenic effect. The limited studies available show no teratogenic effect.

Ammate® has low acute and subchronic toxicity. There is insufficient information to assess its potential as a carcinogen, teratogen, or mutagen.

Atrazine has low acute toxicity when administered orally or dermally. However, it appears to be an eye irritant. It appears to be non-toxic in subchronic studies, and is rapidly eliminated. Although insufficient information is available, atrazine does not show carcinogenic or teratogenic effects in available tests. It does not appear to cause reproductive effects, except when administered at high doses by injection. (Since this route of administration causes moderate toxicity in acute tests, these reproductive effects may be a result of maternal toxicity.) Data suggest that atrazine is mutagenic only after activation by plant enzymes. Mammalian liver enzymes do not appear to be capable of activation.

Bromacil appears to have low acute toxicity, although it may cause mild skin and eye irritation. Limited data suggest that it is rapidly eliminated from mammalian systems. Available data do not allow conclusions to be drawn regarding carcinogenic effect. Available teratogenic studies are negative. Bromacil does not appear to be a carcinogen or teratogen. Some reproductive impairment was noted in one study using bromacil in aerosol form. Bromacil does not appear to be mutagenic.

2,4-D appears to be moderately toxic in acute and subchronic tests, and some formulations appear to be eye irritants. It is rapidly eliminated in mammalian systems. No clear evidence is available that indicates that 2,4-D is a carcinogen, although considerable debate has been generated on the subject and further study is needed. There is some evidence to suggest that 2,4-D causes a weak teratogenic effect; however, the data present no firm basis for conclusion. Most reliable tests indicate that 2,4-D is not a mutagen. Some forms of dioxin have been found as a contaminant in 2,4-D. These forms do not include the form of dioxin (2,3,7,8-tetrachlorodixion), which is known to be highly toxic. Limited information is available on the toxicity of the various forms of dioxin that are found in 2,4-D. One of the forms, 2,7-dichlorodioxin, caused some increased incidence of tumors and reproductive effects; however, the data are difficult to interpret.

Dicamba has low acute and subchronic toxicity, and is rapidly eliminated from mammalian systems. Available tests are inadequate to assess carcinogenicity, teratogenicity, or mutagenicity.

Diquat can be considered toxic by oral, dermal, and inhalation routes. No data were found concerning the carcinogenic potential of diquat. Limited data suggest a possible teratogenic effect. Diquat does not appear to be a mutagen in available tests.

Diuron has low acute toxicity. Slight negative effects (e.g., growth impairment and anemia) have been observed in subchronic effects. No tissue storage occurs, even after chronic administration. Limited data suggest that diuron is not carcinogenic. Although there is some conflicting evidence, most data indicate that diuron is not teratogenic. Diuron does not appear to be a mutagen, although further study is needed.

Glyphosate has low acute toxicity when administered by oral or dermal administration. Moderate toxicity is indicated when administered intraperitoneally. Glyphosate appears to be readily eliminated from mammalian systems. Although no data are publicly available, manufacturer's information suggests that glyphosate shows no potential for carcinogenic, teratogenic, or adverse reproductive effects. Most studies indicate that glyphosate is not a mutagen.

Krenite® has low acute toxicity. A short-term eye irritation has been observed. No data are available regarding the carcinogenic potential of Krenite.® Insufficient data are available to show with certainty that it is not teratogenic or mutagenic. However, since Krenite® was registered relatively recently, it can be assumed that the full complement of tests was conducted and the results have been found to be acceptable by EPA.

Metolachlor has low acute and subchronic toxicity. Data indicate no evidence of carcinogenic, teratogenic, or mutagenic effect; however, no conclusions can be drawn based on the limited data available.

Picloram has low acute toxicity. Slight adverse effects (in organ-to-body weight ratio) have been observed at high doses in subchronic studies. Picloram appears to be rapidly excreted from mammalian systems. Available data do not allow a definitive statement regarding the potential

carcinogenicity of picloram. It does, however, cause benign neoplastic nodules, and therefore should be suspected as a possible carcinogen until it can be shown that these nodules do not progress to carcinomas. An insufficient amount of data are available to show conclusively that picloram does not cause teratogenic or adverse reproductive effects. Most tests indicate that picloram is not a mutagen, although an insufficient number of reliable tests have been conducted.

Tebuthiuron shows moderate toxicity in acute tests. It appears to be rapidly eliminated from mammalian systems. Although no data are available, manufacturer's information states that long-term studies have shown no indication of carcinogenicity, mutagenicity, teratogenicity, or impairment of reproductive performance. More publicly available information is needed; however, since tebuthiuron was registered relatively recently, it can be assumed that the full complement of tests has been conducted and the results have been found to be acceptable by EPA.

Triclopyr has shown slight acute toxicity, while Garlon® formulations have low toxicity in acute studies. Garlon 3A® causes severe damage to eyes. Although no data are available, manufacturer's information states that triclopyr is not carcinogenic or mutagenic, but can cause adverse reproductive effects and is considered fetotoxic. More publicly available information is needed.

POTENTIAL FOR CONTAMINATION OF SURFACE WATERS AND GROUNDWATER BY HERBICIDES

Herbicides, like other chemicals, tend to move with the movement of water, only more slowly. Sometimes the movement of the herbicide is so much slower than the water that the herbicide can be considered immobile. The movement of a herbicide in a particular area is therefore a study of (1) the flow of water in that area and (2) the tendency of the herbicide to move with the water, or instead, to be retained or degraded. Many of the important parameters of herbicide mobility are a function of the particular site under consideration. Also, parameters that are a function of the herbicide (e.g., solubility and speciation) are highly variable among individual herbicides. For these reasons, this discussion will avoid any generalizations about the likelihood of particular herbicides to contaminate surface or groundwater systems. Instead, this section will discuss the

possible routes of herbicide movement, focusing on the factors that increase and decrease the likelihood of that movement. In the first part, emphasis will be given to the site-specific factors that influence movement. The second part of this section will identify some of the important physical-chemical parameters of the herbicides, and will present a summary of the literature reviewed in Appendix II.

Hydrological Considerations

When herbicides first reach the soil they can move downward into the soil (with the infiltration of water) or they can move over the surface with runoff water. These two pathways are competitive; i.e., the more likely a herbicide is to move downward through the soil, the less likely it is to move over the surface.

Subsurface Flow Much of the water that reaches the soil surface moves downward, infiltrating through the upper soil layers. As it moves downward it encounters first an unsaturated zone and then a saturated one, with these zones divided by the water table. (A number of factors affect the potential for herbicides to move downward through the soil profile and laterally at the soil surface. These factors are discussed later in more detail in regard to soil retention and mobility.)

Once the water moves through the soil into the saturated zone it is called "recharge." Subsurface flow moves from areas of recharge to areas of discharge. In a recharge area, the water table can be at a considerable depth below the surface. In discharge areas, the water table is usually at or very near the surface. For any one recharge area, the associated discharge area can be difficult to define. This is because recharge-discharge systems can be both localized and regional. Local systems involve a relatively short subsurface retention time and a nearby discharge. Regional systems involve longer retention times, usually at deep levels, with discharge at considerable distances from the point of entry of the water. Where there is pronounced local relief (a hilly topography) numerous local systems of flow can be produced. Where topographical relief is negligible, subsurface water tends to move in regional systems. The tendency of local systems to develop rather

than regional systems also depends on the depth of the basal boundary of the system. Deeper systems (e.g., deep unstratified glacial tills) encourage regional flows, while shallow systems (e.g., those bounded by unfractured granite) tend to encourage local flows.

The application of a herbicide to a particular recharge area, therefore, can result in a very local discharge (e.g., a thousand meters away) or introduction to a groundwater flow that extends for many miles. Because New England topographic features can vary considerably in a small area, herbicide applications that are only a few meters apart can result in widely different locations of discharge.

Geologic, as well as topographic, considerations can affect the flow of water and herbicide contaminants. Geologic control is exerted by differences in permeability of the underlying stratigraphy. Layers with higher and lower permeability can affect the direction and rates of flow. A conduit with high permeability can thus move herbicides into a regional system of ground water flow even in hilly terrain where otherwise the movement of the herbicide would be dominated by local groundwater flows. On the other hand, low-permeability layers can block the expected downward flow, and move herbicides laterally to discharge areas that are nearer the point of application than would otherwise be expected.

Many other factors besides the topography and geology of an area control the directions and amounts of water that move through subsurface systems. The infinite variety in flow systems created by these factors (as well as the characteristics of the individual herbicides) makes it impossible to assess the potential for groundwater contamination without a site-specific investigation. As an overall generalization, however, local movement of groundwater may be more important than regional movement in regard to the potential for contamination of water supplies by herbicides applied to rights-of-way. Because of their narrow linear form, rights-of-way are unlikely to take up a large percentage of a regional recharge area. Dilution in a large regional system would significantly decrease herbicide concentrations.

Additionally, since some of the herbicides have short persistence times, the retention times of regional systems may result in degradation of the herbicide before discharge. This is difficult to predict, however, since conditions in groundwater systems are not likely to be as conducive to degradation as soil conditions under which persistence times are determined. Additional information on the persistence of individual herbicides and the conditions for degradation in groundwater are discussed below.

Local systems of flow may be particularly likely to occur in Massachusetts, because of a number of conditions that may divert water laterally away from deep regional systems:

1. Much of Massachusetts, like the rest of New England, has pronounced local relief and therefore its groundwater is subject to local topographic control as described above.

2. The topography of many areas in Massachusetts is bedrock controlled, i.e., underlain by bedrock at shallow depths. Bedrock is less permeable than most soils. Because of the lower permeability, water may be diverted laterally at shallow depths.

3. Bedrock can also provide high-permeability conduits for herbicides by means of fractures in the geologic material. These fractures can transport the herbicide in unexpected directions, depending on the orientation and frequency of fractures.

4. Low-permeability layers at varying depths below the surface (called "fragipans") can divert downward movement of herbicides to more localized lateral movement. Fragipans consisting of compacted layers of clay are found in many parts of southeastern Massachusetts, as well as in other parts of the state.

Thus far, the discussion has assumed that once in the ground water flow, herbicides will move with the movement of the water. However, once the herbicide enters the groundwater, it can undergo a number of reactions, including solution-precipitation reactions, changes in speciation, oxidation-reduction reactions, ion pairing or complexation, adsorption-desorption reactions, and microbial degradation. A review of the literature indicates that the last two are the most important mechanisms for retention and degradation of the herbicides discussed in this report. Adsorption involves the binding of herbicides by weak chemical and physical bonds to charged surfaces of colloidal particles (particles of less than 2 microns in diameter) along with surfaces of silica oxides and other materials. This binding removes a certain amount of herbicide from the solution, depending on the amount and type of charge of the herbicide ions or molecules. More information on this process is presented below in the discussion of adsorption of herbicides in soil.

Microbial degradation of herbicides is probably limited in ground water systems. Since the groundwater is not exposed to the atmosphere, oxygen that is consumed in chemical and microbial reactions is not replenished. Microbial oxidation of only a small amount of organic compounds can severely deplete dissolved oxygen resulting in anaerobic conditions. The significance of anaerobic degradation of the herbicides covered in this report is not known. Recent studies have tried to encourage microbial degradation of groundwater contaminants, and have found that such degradation is significant only when nutrients and oxygen are injected into the groundwater flow.

Surface Flow Runoff of herbicides is most likely under conditions that encourage overland flow of water. Factors that encourage runoff include high intensity rainfall events, long slopes with steep gradients, low infiltration capacity of soils, and lack of vegetative cover or other barriers to slow the movement of water. There are two ways in which runoff can transport herbicides: (1) relatively soluble herbicides can dissolve in water moving across the surface of soil, and (2) herbicides

that are adsorbed to soil particles can be transported during the erosion and movement of the soil particles.

The movement of soluble herbicides in runoff is most likely to occur when the first rainfall following a herbicide application is sufficiently intense to exceed the infiltration capacity of the soil. The second type of runoff of herbicides, i.e., transport while adsorbed to soil particles, frequently involves insoluble herbicides generally considered "immobile" (in studies that examine the potential for downward movement). These herbicides are often held tightly by soil particles at the surface of the soil. During a rainfall event of sufficient intensity, these particles can be removed and transported away from the application site. The most important factor in determining the amount of herbicide moved in this way is the velocity of the runoff water. Increasing the velocity by a factor of 2 enables the water to transport particles 64 times larger (Brady, 1974), thereby transporting a considerably larger fraction of surface particles to which herbicides are adsorbed.

Unfortunately, many of the herbicide runoff studies determine the amount of herbicide moved by runoff as a percentage of the amount applied. Typical results indicate that less than 5% of the herbicide is removed from the application area by runoff. These results lack meaning in terms of the concentrations of herbicide contributed by runoff to streams and other surface water bodies. Additionally, these results can be misleading, in that low percentages lost by runoff (e.g. "less than 1% of the herbicide") give an impression that runoff was found to be insignificant. These small percentages can be significant when the application involves a large portion of the drainage area of a single stream.

Runoff water can either infiltrate into the soil (when it slows down and/or reaches a soil whose infiltrative capacity has not been exceeded) or it can be channeled into stream flow. Once it enters a stream, the amount of dilution of the herbicide contamination depends on such factors as the rate and amount of water moving in the stream, as well as on the percentage of the drainage area that received the herbicide

application. Herbicide "sinks" in streams include plant uptake, microbial and chemical degradation, sediment deposit, and volatilization. If stream velocities are high, however, these sinks are not likely to be significant, and the herbicide will move with the flow of water, either in solution or adsorbed to suspended particles.

Once the velocity of the water decreases, as in a pond, lake, or wetland, the sinks mentioned above become more important, and the herbicide is more likely to be retained in the aquatic system. In general, herbicide retention will be greatest in water bodies with greater biomass (e.g., eutrophied ponds and wetlands) than those with less biomass (e.g., oligotrophic lakes). Components of aquatic ecosystems that are important in the retention and degradation of herbicides include:

- Organic matter, both suspended and in sediments, that can retain the herbicide by adsorption or complexation. Organic matter can also assist in microbial degradation by providing nutrients, and by providing carbon sources for cometabolism.

- Suspended mineral matter, which provides sites for adsorption and microbial degradation.

- Plant and animal matter, which can take up and retain herbicides in tissue, circulating these residues from one trophic level to the next,

- Sediments, which provide very large surface areas for adsorption, high microbial populations, and anaerobic conditions (which may favor the degradation of some herbicides and retard the degradation of others).

In regard to herbicide sinks, wetlands are similar to eutrophic water bodies in that they have a high biomass, thick sediments, and large amounts of suspended organic and mineral matter. When flooded, wetland soils often have a thin surface layer (i.e., a few millimeters

thick) that is an aerobic, oxidized state, overlying the remaining sediments, which are in an anaerobic, reduced state. Wetlands that are flooded during only a part of the year represent a particularly complex situation for predicting herbicide retention and degradation, since the sediments can change from an oxidized to a reduced state within a few days after flooding.

Retention/Mobility in Soil

The upper layers of the soil provide the most significant potential for the retention of herbicides. This is due in part to characteristics of the soil matrix (presented below) and in part to the slower velocity of water moving through the soil. The following soil parameters affect the retention/mobility of herbicides:

Organic Matter Because of its large adsorptive capacity, the amount of organic matter in the soil (expressed as a percentage of the total volume of the soil) may be the most important determinant of the fate of herbicides in the environment. As can be seen in Appendix II, most of the herbicides are more mobile in soils low in organic matter (e.g., 1%), and less mobile in soils with high organic content (e.g., 3%). Although the organic matter content of Massachusetts soils varies widely, a common occurrence is a thin (one or two inch) layer of soil with moderately high percentages of organic matter overlying soils with considerably lower organic content. This sharp decline in organic matter near the surface of soils creates conditions that differ from the conditions under which most tests of herbicide mobility are run. Because herbicides are primarily used in agriculture, tests for mobility often use soil that simulates agricultural conditions, i.e., soils that have organic matter which extends deeply into the soil. Herbicide mobility in Massachusetts soils may, therefore, be greater than suggested by field studies available in the literature. (This would not apply to herbicides that are easily retained by organic matter and are applied at rates that do not exceed the adsorptive capacity of the organic matter in the surface layer.)

Organic matter in the soil also has the ability to support microbial populations responsible for degradation of herbicides. All of the herbicides reviewed in Appendix II are degraded primarily by microbial degradation, as opposed to chemical degradation. Generally, higher microbial populations result in shorter persistence times. The organic matter in the soil provides nutrients necessary for maintenance of active microbial populations. Additionally, some herbicides are degraded by cometabolism, i.e., degradation of the herbicides takes place only in the presence of another carbon source, which the organic matter provides.

Soil Texture Soil is made up of particles of various sizes; the relative proportions of particles of different sizes are generally referred to as the soil texture. Gravels, which have a particle size range of greater than 2 millimeters (by the International Society of Soil Science Classification), have a high permeability and allow rapid movement of herbicides and other materials. Sands, which have a particle size range of 0.02 to 2 millimeters, also have a high permeability. Because of the high permeability, lateral movement is less likely to occur than downward movement in both sand and gravel. Silt particles are 0.002 to 0.02 millimeters in size and have a much lower permeability, while clays, with a particle size of less than 0.002 millimeters (2 microns) have the lowest permeability.

Clay layers can act as fragipans, i.e., layers that slow the downward movement of water to such an extent that they divert the water laterally. Fragipans can be found in most parts of the state. In southeastern Massachusetts they can be found 1 to 2 feet below the soil surface, where they may cause seasonal flooding.

Various combinations of these particle sizes result in soil texture classes, such as sandy clays, silty clay loams, and silt loams. "Loam" refers to a mixture of sand, silt, and clay that exhibits overall properties which are characteristic of the particular combination of particle sizes. A sandy loam is a mixture of sand, silt, and clay in which sand is slightly dominant. One of the most common soils in Massachusetts is a fine sandy loam in which fine sand (0.02 to 0.2

millimeters) dominates. Loamy sands, which contain more sand than sandy loams, are also common. When examining herbicide mobility studies available in the literature, it is important to consider whether the soil used in the test contains as much sand as is commonly found in Massachusetts.

Soil texture affects the lateral movement of herbicides as well as the vertical movement. To some extent, movement in these two directions is competitive. In a sandy loam, water can move downward about 72 inches in 24 hours and can spread to a diameter of 26 inches in the same time period. A clay loam, on the other hand, may allow downward movement of water to a depth of only 36 inches in 24 hours, but the lateral spread during that time period may be 48 inches in diameter (Brady, 1974).

Adsorptive Capacity The texture of the soil also affects the capacity of the soil to adsorb herbicides. Particles that are less than 2 microns (clays and some forms of organic matter) are capable of adsorbing herbicide material by weak chemical and physical bonds. As they move through the soil, herbicides adsorb to the charged surfaces of these particles. Soils have varying adsorptive capacities, depending on the amount and type of clay, the amount of organic matter, and pH. The form of organic matter that is most likely to adsorb herbicide material is humus--a dark, amorphous, and heterogeneous organic mass in a colloidal state (i.e., consisting of particles that are 2 microns or less in diameter). Humus is what is left after microbial degradation of a variety of organic materials. Its surface charge is generated by the dissociation of carboxylic and phenolic groups. Herbicides may be adsorbed onto these charged surfaces, or they may become physically trapped in the irregular inner surfaces of the humic material. Herbicides that are trapped in these inner surfaces are more easily removed from the humus than the ones that are adsorbed onto the surfaces.

Clays vary in their adsorptive capacities by the nature and organization of their surfaces. There are three primary types, montmorrilonite,

illite, and kaolinite, with high, medium, and low capacities to adsorb herbicides, respectively. All three are found in Massachusetts, although montmorrilonite is less prevalent here than in other parts of the country. The relative adsorptive capacities of humus, montmorrilonite, illite, and kaolinite can be expressed as a ratio of 20:10:4:1.5, respectively. One of the most important steps therefore, in determining the ability of a particular soil to retain a herbicide is to determine the amount of organic matter and the amounts and types of clay that are present.

pH The acidic or basic nature of the soil solution exerts an influence on the retention/mobility of herbicides in a number of ways. In Massachusetts, soil pH is low, ranging from about 3.5 to 6 (see Appendix I, Chapter 6). At a low pH, some of the adsorption sites are not available to herbicides that enter the soil, even if the herbicides have the appropriate charge. This is because at low pH, ions normally present in the soil are held so tightly that they resist being displaced by the herbicide. The adsorptive capacity of organic matter is particularly affected by pH in this way. Also, at low pH, some components of the soil that contribute to its adsorptive capacity will change from their usual state of being negatively charged to being positively charged. Herbicides that are normally attracted to and held by these surfaces will tend to stay in the solution; other herbicides, not normally adsorbed, will be retained on the charged surfaces.

The pH of soil can also influence opportunities for microbial breakdown of herbicides. Some herbicides are degraded by a variety of microorganisms representing a wide range of tolerated pH values. Others are degraded by specific groups of microorganisms that may have narrower ranges of tolerated pH values. In general, fungal degradation may be dominant at low pH values and bacterial degradation may be dominant at pH 7 and above. Unfortunately, the role of specific groups of microorganisms in microbial breakdown of herbicides in the field is not well understood.

The above discussion has emphasized site specific characteristics that determine the mobility and persistence of herbicides. Of course, the characteristics of the herbicide itself also determine its fate in the environment. Appendix II presents a literature review of the behavior of individual herbicides tested under a variety of different field and laboratory conditions. The summary of this literature review presented below attempts to make some generalizations about each herbicide.

Also presented below is an explanation of the physical characteristics of herbicides that indicate their potential for contaminating groundwater. In Appendix II, the discussion of the mobility and persistence of each herbicide ends with a table of characteristics for that herbicide which indicate its potential for contaminating ground water. The characteristics chosen as indicators are those suggested by the Hazard Evaluation Division (HED) if the Office of Pesticide Programs, EPA, in a memorandum (June 7, 1983) prepared for use by the FIFRA Scientific Advisory Panel. Before presenting the data on the individual herbicides, the following discussion briefly introduces each of the indicators and the thresholds suggested by HED.

Indicators of the Potential for Ground Water Contamination

Water Solubility The amount of material that will dissolve in water may be the most critical information about a herbicide regarding its potential for mobility, since it is a major determinant of how much material will be picked up and carried by water moving through the soil system. Solubility is expressed in a number of ways; the HED memorandum uses parts per million, which for these purposes can be considered equivalent to the number of milligrams of material which can be dissolved in a liter of water. The threshold value suggested by HED is 30 ppm, a relatively low solubility, so the threshold is a conservative one. Most of the herbicides in this report are more soluble than this by one or more orders of magnitude.

Soil Adsorption Coefficient (K_d) K_d, also known as the Freundlich isotherm, or distribution coefficient, is a parameter that indicates the

amount of material which is adsorbed onto soil particles. Indirectly, it indicates the ability of a soil to retain the material. To use the K_d to indicate how much material is retained in soil, the following simple equation can be used:

$$\frac{\bar{V}}{V_c} = (1 + 4K_d) \text{ to } (1 + 10K_d)$$

that is, the rate of movement of ($\bar{V}$) will be faster than the rate of movement of the contaminant (V_c) by a factor of $(1 + 4K_d)$ to $(1 + 10K_d)$. To put it in a more useful way, if $K_d = 1$, then the material will move 5 to 11 times slower than the water or will be retained by a factor of 5 to 11. (Technically, this is true only if adsorption is rapid and reversable, and if the log-log relationship between solute concentration and adsorption is linear.)

The HED threshold of K_d less than 5 is a conservative one, since it implies that the herbicide must move at least 21 to 51 times slower than the surrounding water to be considered as having a low potential for mobility. Some textbooks suggest a K_d threshold of less than 1 as an indication of mobility. This threshold may not be sufficiently conservative, considering the slow degradation rates of some of these herbicides and the amount of water that moves through the surface layers of soil in New England. A K_d value of 3 may be suitably conservative.

This report does not include the K_d value as a mobility indicator, choosing instead to use the K_{oc} value explained below. The difficulty with using a K_d value is that it varies considerably with soil type. An individual herbicide may have K_d values ranging from 1 to 6, depending on the type of soil used in the test. This is due to the fact that the ability of a material to be retained or adsorbed by a soil depends heavily on the amount of colloids (organic matter and clay), among other factors, as dicussed above. Using a soil that is high in organic matter will result in a higher K_d value for a herbicide than using a soil that is low in organic matter. In this report, K_d values have been included where available as part of the discussion of the literature regarding the fate of the individual herbicides in soil.

K_{oc} (K_d Divided By the Organic Carbon Content) In order to factor in the adsorptive capacity of the soil, the K_{oc} is used instead of the K_d. Dividing the K_d by the organic content of the test soil serves to narrow the range of values obtained, thus providing a more distinctive set of values for an individual herbicide. For instance, an herbicide that has a K_d of 6 in a soil with moderate organic matter (2%), and a K_d of 1.5 in a low organic matter soil (0.5%), would have a K_{oc} of 300 in both soils. For the purpose of this report, therefore, it is more reliable to use a K_{oc} value than a K_d value, given the wide variability in Massachusetts soils.

It should be noted that K_{oc} disregards variability in clay content which can contribute to retention of a herbide. The actual retention may be higher or lower than the K_{oc} value suggests, depending on the amount and type of clay present.

The threshold values for K_{oc} suggested by the HED memorandum are conservative. HED appears to have assumed a soil organic content of 1%. Although this is a low organic content, it may reflect conditions in many parts of the state with poor soils. It also may reflect the sharp drop in low organic matter commonly found just below the soil surface in some areas of Massachusetts.

Only a few K_{oc} values, which were determined from actual soil studies, were found for the herbicides discussed in this report. Most of the K_{oc} values in Appendix II were calculated from octanol-water partition coefficients using a method proposed by Hassett et al. (1979).

Speciation An important factor in the ability of soils to adsorb herbicides is the type of charge the herbicide has in the soil solution. If it dissociates in such a way as to form a positively charged ion, it is called a "cation"; if it dissociates to form a negatively charged ion, it is an "anion"; if neither, it is referred to as neutral. Most of the sites available for adsorption in the soil (i.e., the organic matter and the clay) are negatively charged; thus they attract and hold the positive cations. Herbicides that are in the form of negatively charged anions

tend to be repelled by and move quickly past these adsorptive surfaces.

Although soil pH has a modifying influence as (explained previously), the statement can generally be made that herbicides which act as positively charged cations in soil solution are considerably more likely to be held by the soil than either neutral molecules, or anions. Herbicides that act as anions in soil solutions are generally the least likely to be retained by the soil, and therefore are the most likely to be mobile. In accordance with this generalization, the HED memorandum suggests that a potential indication of mobility is the dissociation of the herbicide to form a negatively charged anion.

The speciation of a molecule (i.e., whether it acts as an anion,a cation, or a neutral molecule) is difficult to determine, because most organic molecules can act as two, or all three, different forms depending on the pH of the soil solution. In the preparation of this report, information about the speciation of the herbicides was generally not available in the literature or provided by the manufacturer. To give an indication of the likely speciation, a soil pH of 5 (common to Massachusetts soils, see Appendix I, Chapter 6) was chosen, and then the structure of the molecule was examined for the number of likely sites for the gain or loss of hydrogen protons. After considering the available information on pK_a's for each of the herbicides (pK_a's indicate the pH values at which a change in the amount or type of charge takes place), an estimation was made of the likely species. Where pK_a information was not available, no attempt was made to designate species.

Hydrolysis and Photolysis Half-Lives and Vapor Pressure Compared to the above parameters, these three indicators are of minor importance. The hydrolysis half-life generally estimates the amount of chemical (as opposed to biological) degradation that may occur. The photolysis half-life estimates the breakdown of the herbicide by sunlight or UV radiation. The literature review presented in Appendix II indicates that for almost all the herbicides the primary mechanism of degradation is by microbial action, and that loss due to chemical degradation and

photolysis is insignificant compared to loss due to microbial degradation. Information on hydrolysis and photolysis, however, is included where available.

Volatilization (as measured by vapor pressure) is also not generally significant in determining the total amount of herbicide that can move through the soil. Like photolysis, its importance drops once the herbicide moves into the soil, where the soil spaces quickly become saturated. Movement upward may occur slowly at a rate determined by the volatilization from spaces contiguous to free air above the soil. Lateral movement of herbicides in the soil by volatilization has not been extensively studied, but is considered to be insignificant. The HED memorandum does not provide a threshold for vapor pressure; this report uses a value of 10^{-2} mm Hg.

Soil Half-Life The information conveyed by a soil half-life is not always clear. In a field study, it can represent the dissipation of the herbicide by all routes of loss over time. With a herbicide that is tightly retained by soil, for instance, a half-life measured in the field very likely represents the degradation of the herbicides. For a highly mobile herbicide, the half-life may represent the time required for the herbicide to move vertically or laterally out of the sample site. Laboratory studies are also unclear, because information on losses due to mobility may not be provided.

Because of this uncertainty, and because soil half-lives vary so greatly with soil type and other factors, information on soil half-lives is not included in the list of mobility indicators, but rather is presented in the discussion of available literature. Presenting the soil half-life data as part of a general discussion also allows the description of available parameters such as soil type, moisture, temperature, and pH, which may be important in the interpretation of the half-life.

Summary of Mobility and Persistence Data

Aminotriazole More than other herbicides, the mobility of aminotriazole is dependent on the adsorptive capacity of the soil. This means it can be expected to be mobile in sandy soils, but immobile in soil with high organic matter or high clay content. Aminotriazole has a low to moderate persistence, with half-lives ranging from 6 to 42 days.

Ammate® Limited data suggest that Ammate® may be mobile in soil. Insufficient information is available to estimate persistence, or the factors which affect persistence or mobility.

Atrazine Although conflicting data are available, atrazine can be considered to have low mobility in soil. Factors which increase mobility include low organic matter and higher temperature, moisture, and pH. Runoff of atrazine may occur if a heavy rainfall follows application. Atrazine can be considered a persistent herbicide, with significant residues remaining after 1 to 2 years.

Bromacil Although the mobility of bromacil is significantly affected by the percentage of organic matter, bromacil can be considered highly mobile in a number of soils. Bromacil is a persistent herbicide, with a half-life of 3 to 8 months.

2,4-D The available data do not allow a general statement to be made regarding the mobility of 2,4-D. Important variables seem to be the form of the herbicide (acid, salt, or ester) and soil factors, particularly the amount of organic matter. Surfactants also increase mobility. Most available studies show that 2,4-D is a non-persistent herbicide with a half-life of less than 2 weeks. Monitoring studies have detected 2,4-D in surface water samples. In water, 2,4-D may be stable for several months.

Dicamba Studies show dicamba to be highly mobile in soil. Factors which increase mobility include decreased organic matter and increased pH, although dicamba's high mobility makes these factors less important than they may be for other herbicides. Runoff is not expected to be significant, because of the propensity of dicamba to move downward in soil. The persistence of dicamba in the field is difficult to assess, because of the rapid dissipation which occurs after rainfall. In the laboratory, dicamba has a half-life of 4 weeks.

Diquat Studies show that diquat has low mobility in soil since it is held tightly by clay and organic matter at the surface of the soil. This strong adsorption tends to decrease the rate of degradation. Because of this, diquat is expected to have a long persistence time, although no half-life values are available. In water, diquat is quickly adsorbed to sediments and suspended matter and is taken up by aquatic plants. After being adsorbed by sediments, diquat may persist for several years.

Diuron Available data indicate that diuron is a low-mobility herbicide that stays near the surface of the soil. Lateral movement also appears to be limited. Although conflicting results are available, mobility appears to increase with decreasing organic matter and clay content in soil. Diuron appears to have low to moderate persistence, with residues disappearing after 4 to 8 months. In water, diuron is adsorbed onto suspended and bottom sediments.

Glyphosate Studies show that glyphosate binds rapidly and tightly to soil particles, and has very low mobility. Mobility increases with decreasing clay, organic matter, and increasing phosphate, Na^+ and Ca^{++} concentration in the soil. Persistence is variable (half-lives range from 3 to 133 days), probably due to the different adsorption capacities of soils used the tests. In water, glyphosate appears to be adsorbed to suspended and bottom sediments and slowly degraded by microorganisms.

Krenite® Because of a strong tendency to adsorb to soil particles, Krenite® has a low mobility in soil. Mobility increases with decreasing organic matter and clay content in soil. Because of its tendency to stay near the soil surface, it may be transported by runoff. Krenite® has a low persistence in soil, with a half-life of one week or less.

Metolachlor Available data suggest that metolachlor can be considered highly mobile in soil. Mobility increases with decreasing organic matter in soil. Although no data are are publicly available, a review of registration material by EPA concluded that metolachlor may be persistent in soil (i.e., that it has "a potential for long-term environmental stability").

Picloram Studies show that picloram can be considered mobile in soil. It has a low tendency to adsorb to soil particles. Mobility increases with

decreasing organic matter, with increased pH, and with increasing concentrations of hydrated oxides of aluminum and iron. Runoff studies have indicated that picloram is likely to move in water as it flows over the soil. Numerous studies have shown that picloram is moderately to highly persistent, with half-lives of 1 to 13 or more months. Picloram appears to be less persistent in water than in soil.

Tebuthiuron Studies suggest that tebuthiuron is mobile in the soil. Because of its tendency to move with water, factors which affect the mobility of other herbicides have less of an effect on tebuthiuron. It is very persistent, with half-life values ranging from 4 months to 17 months.

Triclopyr Available data suggest that triclopyr is a mobile herbicide. Mobility increases with decreasing organic matter. It can be considered moderately persistent, with half-life values ranging from 46 to 156 days.

EFFECT ON NON-TARGET ORGANISMS

Herbicide use on rights-of-way affects the ecosystem by removing part or all of the vegetation. The extent of the impact will depend on the selectivity of both the herbicides and the method of application. On railway yards and lines, herbicides with a broad range of target species are "broadcast" over the right-of-way. The effect, therefore, is to eliminate the vegetative community that would have developed there, and to decrease the amount of food and cover that would have been provided to animals by that vegetation.

On utility rights-of-way, herbicides with narrower ranges of effectiveness are applied to small areas and fewer plants. The impact, therefore, will be considerably less than that on railroad rights-of-way. On utility rights-of-way, removal of one component of the vegetative community will give a competitive advantage to other vegetation. For instance, studies have shown that the broadcast spraying of a herbicide that kills broadleaf plants (such as 2,4-D) results in the replacement of those species by grasses, sedges, ferns, and a few herbicide-resistant shrubs (EPRI, 1978). A more selective treatment (one that kills trees but avoids damage to surrounding desirable species) would encourage the spread of the

surrounding species by increasing the available light, nutrients, and moisture. Over time, the continued selective removal of trees will theoretically result in a dominance of desirable species forming a stable vegetative community that is resistant to invasion of trees. This subject is discussed in greater detail below in regard to biological control.

Herbicide treatment on both utility and railroad rights-of-way may kill rare or endangered plants. This is somewhat less likely on railroad rights-of-way, because the harsh conditions of the ballast or yard area generally allow the introduction of only common, hardy weed species. On utility rights-of-way, rare plants are more likely to be found, and their elimination could result away from the unintended movement of herbicide from the target plant. A list of rare plants likely to be found on Massachusetts rights-of-way is provided in Appendix I, Chapter 7, along with a suggested method for inventorying rare plants to allow them to be a marked on maps and avoided by applicators.

A number of studies have been conducted to assess the effects of herbicide use on animal communities on rights-of-way. Brambel and Byrnes (1972) reported a species-specific response to herbicide spraying as shown in Table 6. Squirrels and rabbits seemed to prefer the type of treatment likely to cause the least disturbance to surrounding vegetation, i.e., winter basal treatment. Turkeys, on the other hand, increased dramatically in response to the treatment that caused the most disturbance (i.e. broadcast). No clear negative or positive response to any of the treatments was noted for deer and grouse. All treatments in this study were conducted with 2,4-D and 2,4,5-T between 1953 and 1957.

Another study by Bramble and Byrnes (1982) showed that favorable wildlife conditions developed after a series of herbicide applications. Fruiting shrubs, such as blueberry, huckleberry, blackberry, dewberry, and witch-hazel, provided wildlife with food and cover. EPRI (1978) found that there were no significant differences between old field communities and the plant communities on utility rights-of-way maintained with herbicides in regard to the number of preferred food plants. The same study conducted a songbird census and found that a

TABLE 6

NUMBER OF TIMES COMMON WILDLIFE SPECIES OR SIGNS WERE OBSERVED ON AREAS TREATED WITH 2,4-D AND 2,4,5-T

Treatment	Deer	Turkey	Squirrel	Rabbit	Grouse
Unsprayed	83	0	6	51	12
Winter basal	59	1	11	25	8
Summer basal	53	1	8	12	5
Semi-basal	62	1	6	3	7
Broadcast	45	31	2	8	8

Source: Bramble and Byrnes (1972).

large number of species used powerline corridors for nesting, cover, feeding, and perching.

Herbicides may cause negative effects on animals by altering the chemical composition of the plants. For instance, they may make desirable species less palatable, or undesirable species more palatable (Richter, 1952) as cited by Pimentel (1971). Also, harmful contaminants may be found in plants treated with herbicides. Swanson and Shaw (1954) concluded that Indian grass concentrated more hydrocyanic acid after herbicide treatment. The potential for these and other effects on specific plants depends to a large extent on the chemical properties of the individual herbicide.

The information presented below summarizes the available information on the effect of the individual herbicides on non-target organisms likely to be found on rights-of-way.

Summary of Data on Toxicity to Non-Target Organisms

Aminotriazole appears to be non-toxic to birds and fish, and slightly toxic to bees. Soil microbial activity may be inhibited by its application. Aminotriazole is not likely to bioaccumulate.

Ammate® appears to be non-toxic to birds and fish, although only limited data are available. One study shows it to be non-toxic to deer. Ammate® may temporarily inhibit soil microbial activity.

Atrazine appears to be non-toxic to birds and livestock. It appears to be toxic to some species of fish and non-toxic to others. Some lower aquatic organisms appear to be sensitive to atrazine. In aquatic ecosystems, atrazine decreases the rate of photosynthesis of some algae, and, perhaps indirectly, reduces populations of zooplankton. Atrazine may concentrate to a limited extent in fish, algae, snails, and fungi. Soil microorganisms show variable responses to atrazine.

Bromacil appears to be non-toxic to birds, fish, lower aquatic organisms and bees, although only limited data are available.

2,4-D appears to be non-toxic to birds. Its toxicity to fish and aquatic invertebrates varies with formulation and species of fish. Mammalian wildlife do not appear to be adversely affected by 2,4-D, except in response to changes in vegetation caused by its application. Little adverse effect is observed after exposure of livestock, bees, or soil organisms to 2,4-D. It does not appear to bioaccumulate in a variety of terrestrial and aquatic organisms tested.

Dicamba appears to be non-toxic to birds and livestock. It appears to be moderately toxic to fish, depending on the species. Dicamba appears to be toxic to a number of aquatic organisms, although data are contradictory. Dicamba can be considered non-toxic or only slightly toxic to bees. Limited data suggest that dicamba does not harm soil microorganisms.

Diquat appears to be non-toxic to birds. It appears to be toxic to some species of fish but not to others. Aquatic invertebrates seem to be able to tolerate diquat, except for amphipods, which are very sensitive. Cattle may be somewhat sensitive. Diquat does not accumulate in fish tissue.

Diuron appears to be non-toxic to birds. No information was found on the toxicity of diuron to fish. A number of phytoplankton are sensitive to diuron. One study suggests that diuron inhibits microbial activity in a number of soils.

Glyphosate appears to be non-toxic to birds, although limited data are available. Roundup® appears to be toxic to a number of fish, although it appears to be the surfactant, not the glyphosate, that causes the mortality. The surfactant used in Roundup® is also more toxic than glyphosate to lower aquatic organisms. Glyphosate appears to be non-toxic to bees and soil microorganisms. It does not accumulate in fish tissue.

Krenite® appears to be non-toxic to birds, fish, lower aquatic organisms, bees, and soil microorganisms. It does not accumulate in fish tissue.

Metolachlor appears to be non-toxic to birds and moderately toxic to fish. Limited data are available.

Picloram appears to be non-toxic to birds and toxic to some species of fish. It appears to be non-toxic to lower aquatic organisms, livestock, bees, and soil microorganisms. It does not accumulate in the tissue of livestock or fish. However, it does accumulate in some fungal species.

Tebuthiuron appears to be non-toxic to birds, fish, lower aquatic organisms, bees, and livestock.

Triclopyr appears to be non-toxic to birds, fish, and lower aquatic organisms, although limited data are available on the effect on this last group of organisms.

MINIMIZING THE EFFECTS OF HERBICIDES

As is evident from the above discussion, individual herbicides differ considerably in their potential for impact. The most important way to minimize impact, therefore, is to choose chemicals with the lowest potential for adverse affects. A later section which discusses policy recommendations considers this choice of chemicals in more detail. This section will discuss ways of minimizing impact that apply to all herbicides. Many of these are mentioned in other parts of the report, and are repeated here along with other commonly recommended in the use of herbicides.

- Protective clothing, including gloves and rubber boots, should be worn by applicators. (Goggles or other protective eye-wear should be used when mixing Garlon 3A®, because of its acute eye toxicity). Clothes worn by applicators while spraying should be washed separately from other clothes.

- Containers should be triple-rinsed and disposed of properly. They should never be reused, even after thorough washing.

- Spill contingency plans should be prepared, and the equipment and material necessary for clean-up should be assembled (see Appendix I, Chapter 5).

- Rights-of-ways should be surveyed for sensitive areas including streams, adjacent gardens, playgrounds, and campgrounds. Rights-of-way should be marked, as recommended in Appendix I, Chapter 4, to alert the applicators of the proximity of these sensitive areas.

- A survey of wells (as recommended in Appendix I, Chapter 2) should be conducted, as well as an inventory of rare plants (as recommended in Appendix I, Chapter 7). Applicators should be trained to recognize rare plants they might encounter.

- Access to the right-of-way should be restricted after herbicide application, especially when the areas are known to be frequented by berry or mushroom pickers. (A study should be conducted to determine ways to restrict access, and the appropriate duration of restricted access after spraying various herbicides.) Methods should be developed for marking berry producing plants that are next to target plants receiving treatment.

- Thickeners and other means of controlling drift should be used. In the summer, treatment should be restricted to morning or evening hours to reduce the movement of volatile herbicides.

- To minimize the potential for contamination of ground water and surface waters, particular attention should be given to the choice and use of herbicides in areas that may enhance their mobility. As explained above, these include areas with steep or long slopes, exposed bedrock, or soils with coarse textures or low organic matter.

The suggestion has been made to minimize impact by reducing the frequency of herbicide applications. This idea is worth further study in regard to railroad yards and lines, where herbicides are applied every year. Because of the pending regulatory questions, spraying has been suspended in some areas for the past 1-2 years. During the course of this study, visual inspection of the areas that have not been treated showed some encroachment of vegetation along the sides of the right-of-way. Occasional plants were seen on the ballast near the track. Although no surveys were made, the intrusion of vegetation appeared to occupy less than 5% of the area normally sprayed. This slow invasion of vegetation suggests that applications might be made every two years instead of every year, without jeopardizing the safety of railroad operations. An objection has been raised that the decreased frequency of treatment would allow additional vegetation, including deeper-rooted perennials, to invade, requiring increased amount of herbicides for their removal. However, the amount of herbicides used

every other year would have to be more than twice the usual amount applied, to outweigh the benefit of decreasing the frequency of the application to every other year. Given that railroads currently have a higher rate of application (in pounds of active ingredient) than any use of herbicides in Massachusetts (except highway vegetation control), it is unlikely that this already high rate would have to be increased by more than a factor of 2 to control the additional weeds. Studies comparing the effects of applying herbicides every year and every other year should be conducted to determine the total amounts of herbicides needed over time.

On utility rights-of-way, decreasing the frequency of herbicide treatments is not likely to decrease the total amount of herbicides used over time and may even increase it. On utility rights-of-way, the total amount of herbicides used is determined by the number of trees that invade the right-of-way. Assuming the treatment is effective, each tree receives one treatment. Theoretically, the same amount of herbicide would be used over time if treatments were made--for example every three years or every five years. This assumes that the frequency of application has no effect on the number of trees that invade over time. In fact, visual observation suggests that rights-of-ways that were treated every five to six years (due to budgetary constraints) resulted in a greater number of invading trees than rights-of-way that were treated more frequently (every three to four years). The explanation for this may lie in the importance of competition from desirable species in the inhibition of tree invasion. With the additional growth that occurs between treatments, larger trees take up more light, nutrients, and moisture than younger, smaller trees. Over time, eliminating the trees with more frequent herbicide treatment may decrease this stress on the surrounding desirable species and allow the surrounding vegetation to become increasingly able to resist tree invasion.

PHYSICAL ALTERNATIVES

The alternative to herbicide use in the control of vegetation on rights-of-way is the use of physical means of killing vegetation, including chainsaws, larger cutting machines, and fire. The following section presents each of these and discusses their advantages and disadvantages.

Handcutting

Cutting trees with chainsaws is the most common alternative to herbicide use in controlling trees on Massachusetts rights-of-way. This practice involves severing trees near the base, and then cutting the tree into sections that can be piled nearby. (Sometimes the stump is then treated with herbicides to prevent sprouting). Girdling is another way of using axes or chainsaws. Girdling involves making shallow cuts around the trunk to disrupt the flow of nutrients to the roots. Chainsawing also involves trimming trees that interfere with sight lines or wires on railroad rights-of-way. Most trimming involves removing side branches of trees that extend into the right-of-way area.

The advantage of handcutting is that it avoids introducing chemicals into the right-of-way and the surrounding environment. Another advantage is the degree of selectivity that can be achieved; with careful cutting using small chainsaws, surrounding plants suffer minimum damage. A further advantage is that handcutting prevents contact with wires when trees are at dangerous heights.

The disadvantages of handcutting are:

1. High cost (discussed in more detail below in the comparison of alternative control measures).

2. The failure to control trees that sprout rapidly. Many trees in Massachusetts, including oak (one of the most common) are capable of sprouting from cut stumps. Handcutting of these trees results in an increase in the number of stems per acre.

Because of the rapid growth of these sprouts, handcutting may have to be done every year.

3. The serious hazard to workers. A survey of workers by Vegetation Control Service, Inc. (conducted for this study) noted 36 cuts from chainsaws during the period of 1976 to 1983, based on worker's compensation data for a total of 21 employees. Cuts to the leg, knee, hand, and head numbered 12, 14, 9, and 1, respectively. Causes of chainsaw accidents include loss of balance while using the saw, skidding and bounding of the saw, and extended follow-through of the saw after severing the tree trunk. "Kickback" is also a major cause of accidents. Kickback is the sudden movement of the saw upward and back toward the operator due to some interference with the movement of the chain. Steep slopes are a contributing factor in chainsaw accidents, and handcutting is not recommended on slopes greater than 30%. Wet conditions also increase the likelihood of accidents.

To minimize the hazards associated with handcutting, proper handling of the chainsaw is necessary. Important considerations include:

- Starting and maintaining all cuts at full throttle;
- staying clear of the path the saw will follow on completion of the cut;
- adjustment of the throttle speed so that the chain does not move when the engine is idling;
- starting the saw when it is on the ground (i.e., no "hip starts").

Recent technological advances have resulted in chainsaws that are considerably less likely to kickback. However, it is likely that even with proper handling and new technology, the potential for serious accidents will continue to be high.

Mechanical Cutting

The use of larger machinery to cut vegetation on utility rights-of-way includes:

Pushing, or uprooting the trees with a tracked vehicle equipped with a push bar; the debris can be removed or left for slash disposal. Erosion controls must be used because of the severe disruption of the soil.

Scalping, or scraping off all plants and the top layer of soil. This method can be used only with young trees. Erosion control measures must be used and a considerable amount of valuable soil will be lost. Either wheeled or tracked vehicles can be used. On gentle slopes with few rocks, wide moldboard plows are sufficient. On steeper slopes, the blade should be mounted on a hydraulic hitch to allow raising or lowering. An adjustable bulldozer blade can also be employed if it is used with care.

Discing and plowing, or cutting and turning over vegetation and soil. A variety of conventional tillage implements can be used. This method scarifies the soil, so erosion control measures must be used. Only young trees can be removed by this method.

Rollerchopping, or forcing shrubs and trees to the ground and cutting them into small pieces. Pulled over an area with a tracked vehicle, a roller chopper pushes the tree and cuts it with a blade mounted on heavy metal drums filled with water. Trees up to 6 inches in diameter can be removed by this method. Because it does not intentionally disrupt soil, erosion problems will be less severe with this method than with other mechanical methods. Because the vegetation is cut up immediately, problems of disposal and site appearance will be considerably lessened also.

Sheardozing, or severing all stems close to the ground. Although this method causes soil disturbance by uprooting some vegetation, disturbance can be minimized by waiting until the ground freezes before sheardozing. Stems up to 10 inches in diameter can be removed by this method.

Brushraking and root raking, or scraping up brush and roots and removing cut material. A tracked vehicle is used with a specially designed toothed blade that uproots and removes brush, and a cutting bar attached to the bottom of its teeth to sever roots below the soil surface.

Mechanical cutting on the ballast area on a railroad right-of-way is not possible, because both above and below ground biomass must be killed

without disturbing the ballast itself. Control of trees and shrubs on the side of the track can be done mechanically, however, using a high-rail vehicle especially designed as a brushcutter with large cutter heads mounted on flexible arms that can sever trees up to 14 inches in diameter and cut a swath up to 28 feet on both sides of track centerline.

The benefit of mechanical cutting on both utility and railroad rights-of-way is that it avoids the introduction of chemicals to the right of way while it decreases the danger to the operator imposed by handcutting tools such as chainsaws. The disadvantages of mechanical cutting, however, include other safety problems caused by the use of machines with large cutting blades. The railway brush cutter may be particularly dangerous in this regard. With its 7-foot cutting blades rotating at very rapid speeds, vegetation and other material with which it comes into contact are likely to spin off in all directions. Personnel must walk the tracks along with the vehicle, staying out of range of the flying material but close enough to be able to warn people who may be near the right-of-way. This machine may pose a particular hazard to children in urban areas who may use the railway as a play area, as well as those in rural areas who may use the railway as a path to follow through wooded areas. When a brush-cutter was tried in Massachusetts, area residents complained about the adverse aesthetic impact of the cut area and the lack of privacy resulting from the complete removal of vegetation.

Another disadvantage of mechanical control on both railroad and utility rights-of-way is its failure to control trees that are capable of sprouting from cut stumps. As explained above in regard to handcutting, mechanical cutting results in an increase in density of stems per acre.

On utility rights-of-way, another major disadvantage of mechanical cutting is the lack of selectivity in vegetation removed. Even on rights-of-way with a low density of trees, mechanical cutting results in damage to large areas of vegetation, especially since many of these machines leave a swath of damage as they move from one target tree to the next.

Other problems with mechanical control include erosion of soils after removal of vegetation and adverse aesthetic impact. Erosion problems occur with scalping, plowing, and other methods that disturb the soil.

Adverse aesthetic impact can be expected from mechanical methods; they tend to leave an area looking "bombed out."

Fire

As a control alternative, fire eliminates trees and if done periodically, it tends to maintain a low growth of vegetation. Many species that are desirable on rights-of-way spread by underground stems or roots and are able to tolerate fire. Controlled burning has been tried successfully on rights-of-way in New Hampshire (Dr. David Olson, University of New Hampshire, personal communication, 10/6/83). Several experiments are underway to test the efficacy of burning on rights-of-way at different times of the year.

Prescribed burning is done in four steps: planning, site preparation, burning, and mopping up. Planning involves tactical preparations and notification of appropriate authorities and local inhabitants. Tactical preparations include deciding upon the necessary weather and soil conditions, planning the direction of the burn and the placement of fire lines, notifying back-up forces in case the fire gets out of control, and planning insofar as possible the strategies to be used should the fire escape.

Part of site preparation includes the clearing of fire lines on either side of the right-of-way. Fire lines are strips at least several feet wide that are cleared of all organic material down to mineral soil. A power line right-of-way would probably require one line on each outer edge and periodic lines crossing the right-of-way, so that the area can be burned in blocks instead of in one long strip, which is harder to control. The necessary width of the fire line must be calculated on the basis of the height, density, and moisture of the vegetation to be burned.

A common way to ignite fires is to use a drip torch, which drips lighted fuel onto the vegetation. (Another method which is not recommended is to ignite the burn with a "Heli-Torch," a helicopter used by the Forest Service which drops flaming balls of napalm onto an

area to be burned.) A standard practice is to start the fire at the outside edges of an area, and allow the burn to move towards the center, thereby extinguishing itself due to lack of fuel. To mop up afterwards, workers must return to the site within hours or days after the fire and check for smoldering remains.

Hand-operated weed burners can also be used as an alternative to herbicides on utility rights-of-way. These high-intensity torches, also called flame guns, consist of a fuel tank pressurized with a hand pump connected to a hose, with a steel tube coil and spray plug which serves as a burner head. A small amount of fuel drips into the coil, where it is heated until it vaporizes and produces a flame similar to a blowtorch. The tree trunk is girdled by the flame, destroying the conductive tissue along the perimeter of the trunk. The method has been shown to be effective on white pine, red maple, sugar maple, birches, aspen, red oak, white ash, and shagbark hickory. The advantages of this method are that it can be used year-round, and it is less costly than cutting the tree or girdling it mechanically. It is particularly efficient at treating sprout clumps, since the flame easily wraps around such smaller stems. Young or thin-barked stems are lethally damaged in 10 seconds or less, while larger stems or thick-barked trees require 20 seconds or more. The disadvantages of this method are: (1) it does not control trees that sprout, and (2) it cannot be used in dry weather or under other conditions of fire hazard.

On railroad rights-of-way, controlled burning was used historically to clear vegetation (to reduce the chance of accidental fires from steam engines). A current method for burning ballast areas is to use a track burner, a high-rail vehicle that runs on the tracks and has two arms, about 15 feet long, which extend perpendicularly to the tracks. These arms carry propane torches which burn the vegetation. The torches are so hot that the vegetation itself does not have to carry the fire, so the burning can be done in relatively wet weather. This method requires two or three people on the truck and two or three people to control the fire.

The advantages of controlled burning as a vegetation control technique are that it avoids the use of herbicides and, at the same time, favors the establishment of desired herb and tree species that resist tree invasion. Additionally, prescribed burning at appropriate intervals will reduce buildup of fuel, (i.e., flammable plant material). This can be especially

important in dry, sandy areas, such as Cape Cod. Over the years, both mechanical and chemical control can lead to a build-up of dead plant material on the right-of-way, resulting in a potential fire hazard which would be eliminated by a controlled burn.

The disadvantages of controlled burning include the following:

1. The greatest danger, of course, is the escape of fire. Controlled burning is usually done in large areas surrounded by a buffer of uninhabited land that could absorb a wildfire. The rights-of-way in Massachusetts constitute narrow strips of land, often in urban and surburban areas, without such buffer zones.

2. Power may be interrupted. In some controlled burns, the Forest Service recommends shutting off the power in transmission lines that cross the area to be burned. This prevents electrical discharge between the lines or between the lines and the ground, sometimes caused by the ionized particles in the smoke which can carry the charge.

3. The conditions needed for safe but efficient burning are restrictive. The moisture of the vegetation and soil must be low enough to allow sufficient combustion, but high enough to avoid burning underground rhizomes and soil organic matter. Air temperature and wind patterns must also be appropriate, and the best conditions for smoke dispersal may be the worst for the escape of the fire. Thus, there may be very few times of the year when conditions are appropriate.

4. Aesthetic impacts are another potential problem along rights-of-way that are viewed frequently by large numbers of people. Vegetation does enter burned sites quickly, but even a few months of looking burned-over may create sufficient public disapproval to restrict the use of fire.

5. Air pollution is a problem, as fire smoke contains particulates, nitrous oxides, ozone, carbon monoxide, and gaseous hydrocarbons. The high amounts of particulates can also restrict visibility near highways.

Other Physical Methods

A suggestion has been made to use physical barriers under the ballast on rights-of-way to prevent emergence of weeds. This approach would be ineffective, since airborne seed introduction and build-up of debris from introduced leaves and other organic matter would still allow weeds to develop. Periodic additions of extra ballast has also been suggested, but this method is prohibitively expensive (it costs a minimum of $5000/mile to upgrade ballast) and would need to be done about every four years.

BIOLOGICAL CONTROL ON RIGHTS-OF-WAY

Biological control is an attempt to control unwanted organisms by increasing the populations of their natural enemies and competitors. Based on an understanding of the ecology of the unwanted organism and its surroundings, effective biological control increases the pressures exerted by its natural enemies and competitors, thus keeping the population of the unwanted organism at low levels. The following section examines this concept as it relates to vegetation control on utility and railroad rights-of-way. Appendix I, Chapter 3, provides more detail on a number of topics discussed in this section.

Control by Natural Enemies

Biological control has classically involved the introduction of an antagonistic species, such as the introduction of the nucleopolyhedrosis virus for the control of gypsy moth. For plants, antagonistic species fall into two groups, grazers and disease agents. A suggestion has been made that grazers, e.g., goats or deer, could be introduced to rights-of-way for vegetation control. The difficulties and limitations in this approach are too numerous to enumerate, but include an inability to assure effectiveness, and a substantial cost and effort to keep the grazers on the rights-of-way.

A more practical approach, at least theoretically, is the introduction of plant pathogens. In natural systems, a number of tree species occasionally suffer high loss to wilts and other diseases caused by fungi. Severe dieback of ash has been achieved in nursery studies when the trees are inoculated with a pathogenic fungus. However, even though potentially lethal pathogenic fungi exist for all the tree species likely to be found on rights-of-way, the approach is not yet practical for a number of reasons. One reason is that most tree diseases usually do not kill all, or even most, of their host species. Most host plant species and plant diseases have evolved together to form a stable long-term association. In this association, neither species is likely to eliminate the other in a particular area--either by

destruction of all the trees by the fungus or by complete resistance of the plant to the fungus. Thus, increasing the population of an indigenous pathogenic fungus would not be sufficiently effective in controlling trees on the right-of-way.

It may be possible to introduce a pathogen that has not yet evolved to form a stable association with its host species, and thereby potentially kill most of the trees on a right-of-way. The obvious problem with this approach is that anything that would kill a significant number of trees on the rights-of-way would also kill a significant number of trees in adjacent and surrounding areas. The Dutch elm disease, caused by an introduced fungal pathogen to which the elm had no resistance, provides an example of the rapid spread and large-scale decimation that can occur upon the introduction of a pathogen. Because fungi are easily disseminated in the environment by wind, water, and insects, it is unlikely that the introduced pathogen could be contained within the right-of-way.

On railroad rights-of-way the introduction of antagonistic species is even more limited. The goal of vegetation control on railroad rights-of-way is to eliminate all vegetation. No super-pathogen exists that will kill all species of plants. Conceivably, between 10 and 100 different species would need to be released, increasing the danger of the fungi spreading to adjacent areas beyond the right-of-way.

The suggestion has been made to use a certain kind of fungi that would kill young seedlings and have a wide host range (hundreds or thousands of hosts), as a supplement to using herbicides or other means of control. These fungi (which cause "damping off" of seedlings) could be introduced to the railroad bed to prevent new seedlings from establishing in the cleared area. However, the extensive inoculation, throughout the Commonwealth, of a fungus with such a wide host range is bound to endanger local agriculture. Furthermore, these fungi grow best in moist, crowded conditions, which are not likely to be found on railroad rights-of-way.

Control by competition

Biological control also includes the regulation of unwanted organisms by an increase in the competitive pressures exerted by surrounding organisms. Although this concept is not applicable to railroad ballasts where all species must be eliminated, it may have considerable importance in controlling vegetation on powerline rights-of-way and in brush control on railroad rights-of-way.

Encouraging competition on rights-of-way means promoting and maintaining the growth of so-called "desirable" species, i.e., low growing shrubs and herbaceous plants, to the exclusion of trees. Normally, shrubs and herbs are dominant only for a limited time, being gradually replaced by trees. This "succession" involves a gradual change in vegetative communities, leading eventually to a "climax" community of trees that is able to maintain itself over time. Appendix I, Chapter 3, contains a discussion of the various theories which have been advanced to explain and predict succession, and how these theories may relate to the control of vegetation on rights-of-way. In theory at least, biological control can be accomplished if the pre-climax species (shrubs and herbs) can become a stable vegetative community successfully preventing the invasion of trees.

Stable vegetative communities have been identified by a number of researchers. Niering and Egler (1955) reported a stand of Viburnum lentago in southwestern Connecticut that had no tree invasion for at least 25 years. Niering and Goodwin (1934) identified communities of witch hazel, speckled alder, sheep laurel, and other species in various parts of Connecticut that appeared stable to tree invasion for up to several decades. In another study, a fire line was cleared and harrowed through a southeastern New York forest between 1934 and 1936. In one segment, a complex of stable tree-less communities (including ferns, sedge and, bushes) resisted tree invasion until at least 1953 (Pound and Egler, 1953). Horsley (personal communication, 9/8/1983) described a power line right-of-way in Pennsylvania that had been sprayed with herbicides once 40 years ago, after which a

community of ferns developed. Tree seedlings germinated in the community, but the seedlings did not emerge above the fern cover.

Mechanisms that may be responsible for the resistance to tree invasion in these examples include the following:

1. Soil conditions that are unfavorable to trees but are tolerated by a number of shrubs and herbs; e.g., heaths may inhibit tree invasion by the buildup of acid duff in the soil;
2. Grazing, e.g., deer browsing of young trees;
3. Periodic fires;
4. Allelopathy (the production of chemicals by one plant that are inhibitory to the growth of another plant); and
5. The "head start" provided by a pre-existing or rapid buildup of a high density of shrubs and herbs, particularly those that can spread by underground stems.

Herbicide applications and/or mechanical cutting are often cited as mechanisms to inhibit tree invasion. On the surface this idea may seem to be somewhat circular, i.e., that tree invasion can be prevented by the removal of trees. However, it appears that tree removal may give a competitive advantage to desirable species by decreasing the moisture, light, and other stresses imposed by the trees. Over time, this may promote an increasing density of desirable species, which may in turn result in a slow decrease in the invasion of new trees. Appendix I, Chapter 3, presents examples in which periodic chemical treatment significantly decreased tree invasion over time.

A number of species commonly found in Massachusetts have been found to be components of stable shrub communities. A brief discussion of a number of these species, including sheep laurel, witch hazel, blueberry, goldenrod, little bluestem, and others is included in Appendix II, in Chapter 3. A common characteristic of these plants is an ability to grow in a variety of conditions including low-nutrient soils and wet or dry habitats.

In the preparation of this report the following treatments (and their limitations) were identified that may be used to promote the growth of desirable species and limit tree invasion:

1. Planting indigenous species Some species are limited in abundance by a low density of seeds, and would benefit by the addition of seeds to rights-of-way. Blackberry, for example, can form a dense cover following disturbance if its seeds are present in sufficient numbers. If there are only a few seeds, it can take much longer to reach a high density (Bramble and Byrnes, 1982). These authors make a more general statement that species which produce abundant seeds at the right time (for example, witch hazel which produces seeds in the fall) often become prominent in the stable shrub communities.

Experts disagree on the efficacy of planting along rights-of-way. Egler and Foote (1975) state that this approach is impractical because of the amount of care that would be needed to maintain the introduced plants. On the other hand, Littlefield produced a list of species appropriate for planting on rights-of-way, including viburnums, bristly locusts, hawthorns, hawkweed, fireweed, and ferns (L. Littlefield, personal communication 10/3/83). Tilman (1976) planted several species of plants on a southeastern New York right-of-way.

Because of the expense involved, it would probably not be practical to introduce whole plants. Introduced species should be restricted to those that can be seeded and which do not require soil manipulation or great care. The simplest technique is to broadcast seed (perhaps little bluestem) along a right-of-way during the time in which trees are being mechanically or chemically removed. More intensive plantings might be possible in small, ecologically sensitive areas as an alternative to mechanical or chemical control.

2. Soil amendments A logical approach would be to amend the soil to make it suitable to desirable species. However, there are several difficulties with this approach:

- Soil treatments that are of benefit to shrubs and herbs are often of benefit to trees as well. For example, fertilization to benefit bracken fern may also encourage oak.

- Often, the principle components of stable shrub communities are species that tolerate poor, acid soils. These species might lose their dominance if soil conditions were "improved" by additions of fertilizers or lime.

- Soil treatments that favor one set of desirable species may hinder another. For instance, some desirable grasses, such as little bluestem, can be encouraged by liming. However, liming can damage several desirable shrubs such as blueberries, mountain laurel, and sweetfern, which prefer acid soils.

3. Use of symbiotic fungi Certain kinds of soil fungi called "mycorrhizae" form symbiotic association with plant roots. In these associations, the plants provide the fungi with simple sugars while, in return, the fungus provides the plant with the phosphorus that it absorbs from the soil. Plants that enter into mycorrhizal relationships are often able to grow faster and out-compete plants that are not associated with mycorrhizae. To some extent, different types of mycorrhizae are limited in the hosts that they can infect. Three types of mycorrhizae are of interest in regard to rights-of-way:

TYPE OF MYCORRHIZAE	ASSOCIATED PLANT SPECIES
Ectomycorrhizae	Oak, beech, alder, pine, spruce, hemlock.
VA (vesicular-arbuscular)	Maple, cherry, sassafras, ferns, most herbs and shrubs.
Ericoid	Blueberry, huckleberry, laurel, azalea, rhododendron.

Since VA and ericoid mycorrhizae infect primarily desirable species (except for maple, cherry, and sassafras) it is conceivable that

desirable species could be given a competitive advantage by increasing the concentration of these mycorrhizae. At present, however, it is not possible to produce large enough quantities of innoculum of VA or ericoid mycorrhizae to consider adding these to the soil at levels above those that naturally occur there. Abbott Laboratories and Monsanto Chemical Company are both attempting to produce large amounts of VA-mycorrhizae, but costs are prohibitive. For example, a 1-quart container of spores (in soil), sufficient to cover less than 100 square feet of soil surface, costs approximately $25. Another limitation is the lack of information regarding application techniques that will ensure the success of the mycorrhizae in the field.

4. Fire Many species that may be able to resist tree invasion are favored by fire. These species, which include little bluestem, sweetfern, huckleberry, and sheep laurel, tend to appear on burned-over sites soon after a fire. Many of these species are colonial, that is, they spread by underground or above-ground stems or roots, which partially explains their ability to remain alive during fires and to sprout afterwards. One way of encouraging species resistant to tree invasion, therefore, may be to periodically burn the right-of-way. The benefits and limitations of controlled burning have been discussed previously in this report. The conclusion of that discussion was that the hazards associated with burning limit its use on rights-of-way. Research in this area is currently underway, however, and the possibility exists that controlled burning may yet be a means for encouraging desirable species as well as removing trees.

5. Planting commercial crops So far, this investigation has evaluated treatments from the point of view of minimizing cost and effort. However, use of the right-of-way to grow commercial species can also be considered a form of biological control. Utility rights-of-way can be used for pasture, farmland, or wood production. Blueberries, for instance, may be grown in areas with acid soils. Wood production can include trees grown for boards and Christmas trees, and for reconstituted products, including paper and particle board. A pilot study on wood production along Maine highway rights-of-way indicates

that such an effort could be profitable within a few years (Hatton, 1982). On utility rights-of-way, disadvantages of growing commercial crops include the logistical problems of access and that, for some crops, the use of chemical control agents would be greater than that currently used.

Conclusions Regarding Biological Control

A number of reviewers of the draft GEIR felt that the report was not sufficiently enthusiastic about biological control. The draft concluded that:

1) Biological control is not a viable option for maintaining a vegetation-free area on railroad rights-of-way. A vegetation-free area is not a natural biological occurrence, and therefore it is difficult to find and exploit a biological mechanism for its maintenance.

2) A greater chance of success for biological control may be possible when considering powerline rights-of-way. Competition from desirable species should be encouraged as a biological mechanism for decreasing the need for biological control. Selective removal of undesirable species, by either chemical or mechanical means, can be supplemented by the addition of seeds of desirable species, improving soil conditions, and growing commercial crops where conditions permit.

The above statement regarding biological control on powerlines should have been more strongly worded in spite of frequent admonitions throughout the report to use competition from desirable species as the basis for any control effort on powerlines. This report strongly recommends biological control when it is defined in this sense; i.e., as control by competition. The term "biological control" was not used because this approach usually is based on the use of non-biological methods; that is, the selective chemical or mechanical elimination of the undesirable species as the means to achieve control by competition; and use of the term is therefore confusing, and "control by competition" or "encouragement of desirable species" should be used instead.

Again, this report strongly recommends a biologically based control of undesirable vegetation on utility rights-of-way by competitive pressure exerted by desirable species. It also recommends the elimination of undesirable species by chemical or mechanical means as an important element in achieving control by competition. Once this is achieved, further need to eliminate undesirable species by mechanical or chemical means should be minimal or nonexistent. This biologically based approach should be an integral part of both utility and railroad (brush control) efforts.

INFORMATION REQUESTED BY REVIEWERS

The review of the draft GEIR resulted in a number of requests for additional information. Some of that information has been integrated into other sections of this report. This section presents the remainder of that information as a series of short discussions.

Additional Information on Utility Practices

Information on the practices of the three major utilities (which maintain about 90% of rights-of-way in Massachusetts) is presented in the following tables. The first table shows the active ingredients used, the types of application, and the mixtures used. The second table, provided by New England Power Co., indicates which active ingredients are potentially usable against which trees.

Railroad Practices

Additional information was requested on the vegetation control practices of railroads; namely,

1) How many applicators operate in Massachusetts? Two applicators control vegetation on railroads in the Commonwealth: Railroad Weed Control Inc. of Westfield, Massachusetts, and Asplundh, Inc., of Willow Grove, Pennsylvania.

2) How many track miles are treated with herbicides? In 1983, 780 miles were identified as needing treatment, 480 miles were treated. The remaining 300 miles were not treated, due to proximity to water bodies.

3) What is the frequency of treatment on various segments of track? All track in Massachusetts is treated on an annual basis.

4) What mixtures of herbicides are used, and how often are they used? Mixtures are used in 100% of ballast control efforts. Preemergent treatment, done on 65-70% of the track miles, involves mixtures of atrazine and diuron. Postemergent treatment, done of 30-35% of the track miles, involves mixtures of atrazine, diuron, glyphosate, and sometimes metolachlor. Brush control is often limited to the use of glyphosate alone.

5) How often is manual or mechanical control used, and what railroads use this type of control? No manual or mechanical control is done on

UTILITY PRACTICES

	New England Power Company	Northeast Utilities	Boston Edison Company
Active ingredient used and type of treatment	Krenite - foliar Tordon 101 (2,4-D and picloram) - foliar Tordon RTU (2,4-D and picloram) - cut stump Garlon 3A (triclopyr) - foliar Garlon 4 (triclopyr) - selective basal Roundup (glyphosate) - foliar	Triclopyr - basal Dicamba - cut stump Ammonium sulfamate - cut strip Krenite - stem foliar	Krenite - foliar Tordon 101 (2,4-D and picloram) - foliar Triclopyr - basal Tordon RTU (2,4-D and picloram) - cut stump
Acreage* covered by:			
backpack	75%	1-2%	100%
hydraulic sprayer	25%	96-98%	15%
manually cut	5%	1-2%	15%
Mixtures used:	Tordon 101 and Garlon 3A (2,4-D and picloram), and triclopyr	none	Tordon 101 (2,4-D and picloram) Tordon RTU (2,4-D and picloram)

*Acreage can total more than 100% because some areas are treated by more than one method.

HERBICIDE EFFECTIVENESS

Tordon 101:	Ash - Resistant Birch - Effective Conifers - Effective	Maple - Effective Oak - Moderately effective
Tordon RTU:	Ash - Moderately effective Conifers - Unnecessary (except for Pitch Pine)	Maple - Moderately effective Oak - Effective
Banvel 720:	Ash - Effective Birch - Moderately effective Conifers - Moderately effective	Maple - Moderately effective Oak - Moderately effective
Banvel 520:	Ash - Effective Birch - Moderately effective Conifers - Effective	Maple - Effective Oak - Moderately effective
Banvel CST:	Ash - Effective Conifers - Unnecessary (except for Pitch Pine)	Maple - Moderately effective Oak - Effective
Fosamine Ammonium:	Ash - Moderately effective Birch - Effective Black Cherry - Ineffective Black Gum - Ineffective Conifers - Moderately effective	Junipers - Ineffective Maple - Moderately effective Oak - Effective Sassafras - Ineffective
Glyphosate:	Ash - Effective Birch - Effective Conifers - Moderately effective	Maple - Effective Oak - Effective
Triclopyr:	Ash - Effective Conifers - Ineffective	Maple - Effective Oak - Effective

ballast by any railroad in Massachusetts. Large (e.g., Conrail) and small (e.g., Providence and Worcester) railroads use both manual/mechanical controls and herbicides to control brush. No clear information is available on the amount of brush control that involves only herbicides or only mechanical or manual effort; responses to questions in this regard were that herbicides are often used as a followup treatment after mechanical/manual control.

6) Is the use of fire currently an alternative in Massachusetts? Apparently, there has been no use of ballast burners in Massachusetts, although equipment is available. The equipment is similar to highrail vehicles used for herbicide application, with propane torches in place of sprayers. These torches provide a very hot, quick burn which is not likely to smoulder and spread to adjacent areas. Cost data were not available, although railroad sources stated that costs would be "prohibitive." The National Railway Association could not name any manufacturers of this equipment nor any railroad that used it. The National Railway Association stated also that control by fire could not be used in any state which prohibited open burning, including Massachusetts.

7) Why are such high rates of herbicide application used in vegetation control on ballasts? No data were found that justified the use of current rates of application. The need for such rates is highly questionable.

Highway Practices

A number of comments on the draft GEIR requested additional information on use of herbicides on highways; specifically, those comments from

MEPA
Department of Public Health
Town of Belmont
Cambridge Water Board
Massachusetts Association of Conservation Commissions
Nashua River Watershed Council
Cape Cod Planning and Economic Development Commission

The process by which decisions are made at MDPW regarding vegetation control is as follows. Each of the eight Districts of the MDPW has a district road-maintenance engineer who inspects highways for

maintenance problems. This person identifies areas that need treatment, specifies the type of control (e.g., manual cutting or a specific herbicide) that should be used on that particular vegetation problem, and estimates the cost for the treatment problem. The Maintenance Division at MDPW headquarters reviews these requests and allocates portions of the total MDPW funds available for vegetation control to the individual districts. Since materials for vegetation control are purchased by the Maintenance Division before the start of the application season, it is likely that there is an estimation on the amount of treatment and the herbicides to be used on a statewide basis with input from the districts.

An inquiry was made at MEPA's request into the status of aminotriazole (including Fenavar®, which contains aminotriazole) at the Massachusetts Department of Public Works (MDPW), given the evidence of carcinogenicity presented in the draft GEIR. The Maintenance Division of the MDPW stated that it would not be used in the next fiscal year (summer of 1985) because of the decision on the part of EPA to restrict the chemical (thereby requiring applicators of aminotriazole to be certified in right-of-way maintenance).

Prometon

Although originally considered for inclusion in this report, prometon was dropped from the list. Ciba-Geigy is not marketing prometon for right-of-way use since it recognizes there are more cost-effective alternatives. According to a company spokesman, prometon has not been used for right-of-way use in Massachusetts for a considerable time, and they do not expect any significant use in the future. It is still registered for use on rights-of-way.

Outline for Long-range Management Plan

The following is a brief outline of the important steps in assuring minimal need for vegetation control when creating a new right-of-way.

The utility plan presents the important activities of the first 10-12 years. The railroad plan takes a different approach, discussing materials which could be added to the ballast upon creation of the right-of-way, or with more difficulty, at a later stage. The ideas presented for utility rights-of-way were generated by the author in a previous study (Arthur D. Little, Inc., 1979). The ideas presented for railroad ballasts, however, were generated by the author without benefit of sufficient previous study. No written material or current research was located on the ideas presented.

Utility rights-of-way and brush control along railways

Purpose: To increase competitive pressure exerted by desirable species and thereby decrease the need for vegetation control.

Methods: 1) Maximize sunlight penetration (most undesirable species are shade-tolerant, whereas desirable ones often are not); 2) chemically kill roots of any undesirable trees capable of sprouting; 3) seed or plant desirable species or increase their density through soil manipulations.

Plan: 1) Overall approach is to give the desirable species a good start by paying close attention to the right-of-way in the first five years. After 10 or 12 years, the right-of-way should approach stability and require minimal effort because of a low density of undesirable species.

Specific Approach: Year 1: Clearcut to maximize sunlight penetration.

Year 2: Chemically treat stump of any trees capable of sprouting.

Years 3-5: Deplete seedbank of undesirable trees by cutting or spraying (should be done yearly or every other year to prevent seedlings from shading desirable species). Also seed, plant, or otherwise encourage desirable species.

Years 6-12: Treat every three years to deter invasion of exogenous seeds. Continue to chemically kill roots of sprouting trees.

Year 12 Onward: Monitor and cut or treat every three to five years as necessary. Need for control (stems/acre) should decrease slowly.

Ballast of railway

Purpose: To deter plant invasion, to prevent soil buildup, and to remove plants as they appear.

Methods: 1) Generate inhospitable conditions for plants by adding materials to the surface of the ballast which increase stress on plants; 2) decrease the rate of soil generation through the addition of materials which deter microbial degradation; 3) remove vegetation manually or by fire or chemical treatment as necessary.

Plan: The overall approach is to discourage seeds from germinating on the ballast. It is important to prevent the plants from invading so that subsequent soil buildup does not occur. Soil buildup greatly accelerates the further invasion of plants by providing favorable sites for seed germination. Currently, this preventative effort has been based on the use of preemergent herbicides. The alternative suggested here is to prevent invasion by making the ballast more inhospitable; i.e., more stressful to plants, and by decreasing the rate of organic decomposition which precedes soil buildup. If vegetation appears, the vegetation would be removed and additional efforts to slow organic decomposition would be made.

Specific Plan: Add materials to the ballast which would increase plant stress, such as a) adsorbants and chelators, e.g., activated charcoal and EDTA, respectively, to compete for nutrients; b) absorbants, e.g., silica gel particles, to compete for moisture; c) any black material, e.g., low-grade coal, to increase surface temperatures. Closely monitor the ballast for plant invasion and remove as necessary.

The most effective means of removal would be manual, if care were taken to remove as much of the root system as possible. While costs for manual removal would be prohibitive if done by the railroads themselves, cooperative agreements with towns to maintain the track acreage within that town could be a source of economical labor. A number of such agreements have recently been made between towns and utilities to maintain powerline rights-of-way. Particular attention must be paid to the issue of safety when considering similar arrangements on railroad rights-of-way.

If manual removal was not possible, treatment with fire or herbicides would be necessary. If herbicides were used, it is possible that spot treatments may be sufficient. Instead of using a conventional sprayer which distributes material across the entire track, spot treatments could be done as they are on utility rights-of-way; i.e., applied by individuals using sprayers connected to a vehicle carrying herbicide. At a place needing treatment, one or more individuals could direct the sprayers at the invading vegetation. Since most of the invading vegetation encroaches slowly from the sides of the ballast, selective treatment of these areas may alleviate problems which frequently trigger a treatment of the entire ballast.

Because removal by fire or herbicide would leave much of the organic material remaining, efforts must be made to slow organic decomposition leading to soil buildup. Some of the stress-inducing agents (adsorbants, chelators, heat absorbers, etc.) would also deter microbial activity. Additional deterrence should be provided by adding a slow-release bacteriastatic material similar to those in many household products. This should be done along the entire ballast, even in areas without plant invasion, because of the buildup of leaves, spores, and other organic material deposited on the ballast.

The long-range plan would be to add more stress-inducing and bacteriastatic material, and remove vegetation only as necessary. The frequency of these various activities is difficult to assess because this approach has yet to be tested. Several years of testing will be necessary to see if this approach; i.e., eliminating the need for preemergent herbicides by making the ballast less conducive to plant invasion and by decreasing organic decomposition, will be an effective means of controlling vegetation.

Synergism

A number of reviewers expressed concern about the interactions of herbicides with each other and with other man-made compounds. Those who expressed such concern included:

Department of Public Health
Towns of Belmont, Somerville, and Plainville
Massachusetts Association of Conservation Commissions
Vietnam Veterans of America
University of Massachusetts Cranberry Station
Citizens for Safe Use of Pesticides
Citizens Pesticide Council
Massachusetts Audubon Society
Goodwin, Proctor, and Hoar
Lindsay Martuci
Michael Rosebury

The toxicity of two chemicals used in combination can sometimes be roughly predicted from the relative toxicities of each alone. The acute oral LD 50 of Tordon 101, for example, is 3080 mg/kg in rats. The LD 50's for 2,4-D and picloram, individually, are 375 mg/kg and 8200 mg/kg. Such an interaction is termed 'additive.' Sometimes, however, the interaction results in an unexpected increase or decrease in toxicity. An increase in toxicity occurs when an interaction is 'synergistic,' a decrease in toxicity occurs when an interaction is 'antagonistic.'

Predicting the result of exposure to more than one chemical is difficult because of the complexity of possible interactions within the body. When the body is exposed to a chemical, a series of chemical reactions controls the amount and rate of input to the body, transport and storage within the body, metabolic breakdown, excretion, and the adverse effect at the target site, if any. Two chemicals may interact synergistically or antagonistically when they affect the same set of reactions at any of these stages of response. The magnitude of the synergism depends on the importance of that set stage of bodily response as a limiting factor in the toxicity of either of the chemicals. For example, if one chemical is limited in its effect primarily by its inability to pass through the skin, and another chemical increases the absorptive capacity of the skin, a significant synergistic effect may be observed. However, if the first chemical was limited in its effect by enzymatic degradation, something which increased the absorptive capacity of skin would have much less of an effect.

One of the difficulties in predicting interactive effects is due to a change in limiting factors associated with changes in age, sex, and

physiological condition of the organism. Thus, at one time, the organism may be able to metabolize a herbicide, while at a more advanced age, without a full set of necessary enzymes, the organism must rely on tissue storage for protection. At this later time, a synergistic interaction could result from an exposure to a second herbicide competing for tissue-binding sites.

A frequent type of interaction occurs when one herbicide affects the enzymatic activity responsible for metabolizing another. Furthermore, this enzymatic activity is often the limiting step in determining the response of an organism. These interactions can be particularly difficult to predict, since:

1) either chemical may increase or decrease the numbers and kinds of available enzymes,

2) the enzymes affected may either detoxify or activate one or both herbicides,

3) the sequence and timing of the exposure to the herbicides can determine which enzymes are affected and whether they are increased or decreased by the interaction.

This last complication arises in part from 'induction' of enzymes; i.e., exposure to a herbicide may trigger the induction of enzymes which are capable of metabolizing it. If the induced enzymes are non-specific (e.g., the hepatic microsomal oxidase enzymes, which are important in the oxidation of a wide range of foreign compounds), the organism may more quickly metabolize another herbicide upon exposure. In this case, the interaction would be antagonistic. Timing is critical, however, since induction of the important enzymes may not occur until hours or days after exposure to the first chemical, or only after chronic exposure. Timing can also be important when one compound inhibits an enzyme after an accumulation of metabolic products. Necessary enzymes may not be available upon subsequent exposure to another chemical. Lastly, if the exposure is simultaneous, competition for existing enzyme binding sites can occur, leading to a temporary increase in toxic response if the enzyme system is overloaded. The effect in this case will depend in part on the relative affinities of the two chemicals for binding sites.

Interactions which occur at the target receptor (i.e., the cell which ultimately is harmed by the toxin) are less common than those which are involved in absorption, distribution, and metabolism. At the target receptor, many interactions are competitive, since the harmful effect of various toxins is often similar. The interaction, therefore, is commonly antagonistic. Exceptions can occur when two compounds act differently on a single system. A well-known example is the exposure to organochlorine insecticides, which cause hyperexcitability and increase the neurotransmitter acetylcholine, and organophosphate insecticides, which increase the acetylcholine by preventing its degradation by cholinesterase.

The effect of herbicides on sensitive populations can be considered a type of (potentially) synergistic interaction. Instead of interacting with another chemical, however, the herbicide interacts with conditions of the body which increase its susceptibility to the herbicide. Sensitive populations may include people who are ill, taking medication, pregnant, old, dieting, malnourished, and those with genetic traits that impair their ability to tolerate foreign compounds. Some reviewers suggested that all humans should be considered members of a sensitive population. Since the limiting mechanisms which enable the body to tolerate individual chemicals are not well understood, and because these limiting mechanisms may change with the individual and his physiological condition, the suggestion to consider all humans as sensitive has merit. However, the need still exists to consider a category of people who may be more susceptible to herbicides than others.

Effects of herbicides on sensitive populations has received little attention. Most toxicological studies are designed to test the effect of a chemical on normal, healthy organisms. A great deal of attention is given to assuring that laboratory organisms are not in a weakened state which might predispose them to show a harmful effect. The most conservative test for toxicity, however, would use subpopulations which are particularly vulnerable to harm. These subpopulations would have to be defined for each chemical based on the particular response of the organism to that chemical. Sensitive organisms would be those that had:

1) an increased likelihood of absorbing the herbicide,
2) a decreased ability to detoxify or excrete the compound,
3) an increased sensitivity of the receptor site, and
4) a decreased ability to tolerate the effect at the receptor site.

Unfortunately, the number of physiological states which would contribute to these sensitivities are too numerous to test. At best, a thorough risk assessment will identify the numerous diseases or genetic impairments which involve the receptor site affected by the particular compound being studied.

Any information located in the course of this study on the potential synergistic interactions of the fourteen herbicides and their effects on sensitive populations is included in Appendix II.

Degradation

A number of reviewers stated that insufficient information was provided on the degradation of herbicides.

Degradation of herbicides proceeds by a variety of mechanisms, such as microbial or chemical degradation and photolysis. Microbial degradation is probably the most important in the breakdown of the fourteen herbicides addressed in this report, especially once the herbicides reach the soil.

Bacteria and fungi in the soil will break down the herbicide molecule in a series of steps which often begins with such simple changes as the removal of a hydroxyl group and proceeds to more difficult steps such as the cleavage of a ring structure. Numerous microbial species are usually involved in the breakdown of a particular herbicide. A variety of species are often available which can perform the first, and easiest, breakdown steps. As the steps become more difficult, degradation becomes slower and more dependent on the availability of particular types of microbes, and thus on the particular conditions of the soil. Ideally, degradation proceeds until the products are those which are

ubiquitous in nature, such as carbon dioxide and water. 2,4-D, for instance, breaks down completely in a variety of soils into products like succinic acid normally found in those soils. Most herbicides probably do not break down this completely, although few have been studied as thoroughly as 2,4-D. More likely, degradation proceeds until microorganisms capable of degrading them further are not available and the residue, called a terminal residue, remains as is for a considerable time in the soil. One common fate of these terminal residues is incorporation into soil humus or even into the biomass of the microbes themselves.

In the water, microbial degradation occurs in both the water and on suspended particulate matter. Since microbial populations in water and on suspended matter differ, the rates of microbial degradation will depend to some extent on the amount of particulate material and the tendency of the herbicide to be adsorbed to the material. The amount of dissolved oxygen is also important, because it determines to some extent the microbial populations that will be present in that habitat. It also determines the type and rates of enzymatic activity since the activity of many enzymes depends on oxygen availability. Finally, dissolved organic matter, such as humic acid or petroleum contaminants, is also important because it can change the solubility and availability of the herbicide. Increased solubility and availability may be kinetically more desirable since the microbes have access to higher concentrations. However, concentrations can increase to levels which are toxic to the microorganisms.

In both soil and water, the ability of microorganisms to degrade a chemical depends to a large extent on the structure and complexity of the molecule. There are a few rough 'rules of thumb':

1) Aromatic hydrocarbons (i.e., those with rings) are more difficult to degrade than compounds made up of chains, such as alkanes.

2) Branched chains are more difficult to break down than straight chains.

3) Within the aromatics, benzene, a single unsubstituted ring, is more difficult to degrade than polycyclic compounds (more than one ring) or substituted rings (with radicals such as hydrocarbon chains

attached). Addition of chlorine significantly decreases the biodegradability of any ring structure.

The degradation products formed by microbial metabolism or other forms of decomposition may be substantially different from the parent compound, or very similar. More importantly, they may result in an increase or a decrease in toxicity. Photodieldrin, a product of both photo- and microbial degradation of dieldrin, is a well-known example of a degradation product which is more toxic than the parent compound. Also, metabolic products can be more toxic than the original compound after it is activated by enzymatic activity.

As shown in Appendix II, there are often several degradation products which have been identified for a particular chemical. The toxicity of many of these degradation products has not been studied. Additional information in this regard was located during the preparation of the final GEIR and has been included in Appendix II.

Drift

Some comments on the draft GEIR requested that additional information be provided on the potential for herbicides to drift. Specifically, the groups that made this request were:

DEQE Office of Research & Standards
Natural Resources Commission
Smithsonian Institute of Environmental Research
Goodwin, Proctor, and Hoar
Towns of Southampton, Westport, and Plainville

Drift is the movement of herbicide by air currents to locations outside of the target area. Drift has always been a concern in the application of herbicides because of the potential injury to plants outside the local area and because of the potential harm to humans and other organisms.

An important factor in assessing the potential for drift is the particle size of the herbicide droplet. Droplets above 500 microns are generally considered drift safe. Using large droplets, however, may result in

increased amounts of herbicides applied to the rights-of-way, since smaller droplets facilitate greater coverage per volume of herbicide, especially in foliar applications. Application equipment often allows the user to choose the desired droplet size, to the extent that a small stream of herbicide may be released, rather than a spray. Thickeners in the tank mix also contribute to the creation of large droplet sizes. It should be noted that a range of particle sizes is likely to be released by most equipment, even at settings which increase particle size. The proportions of different particle sizes, however, will vary.

The vapor pressure of the herbicide and its carrier is particularly important, since it partly determines the decrease in size of the particle as it moves away from the target area. A volatile herbicide 10 um in diameter will take hours to evaporate completely, while a nonvolatile herbicide of the same size would take months to evaporate. The vapor pressure of the carrier is also important, since it determines to a large extent whether the carrier moves with the herbicide or independent of it. Highly volatile carriers such as kerosene may volatilize quickly and move large distances in vapor form. Detecting the carrier by smell therefore may or may not mean that the person is also in contact with the herbicide. That person would be in contact with both the herbicide and the carrier if 1) the exposure included droplets as well as vapors, 2) the vapor pressure of the herbicides and the carrier were similar, or 3) the evaporation of the carrier reduced the particle size of the mixture to such an extent that it was entrained in the air and carried with the vaporized carrier.

Other important factors include weather conditions and types of applications. The most important weather condition is probably wind, including its speed, direction, and turbulence. Temperature is also important, since volatility increases with temperature. Humid conditions, on the other hand, tend to decrease volatility. Sunlight can make a difference if the herbicide is susceptible to photolysis, since photolysis can decrease the concentrations in the air as well as on leaf surfaces where further evaporation could take place after application.

Types of application are also important. Drift is most likely in foliar application where the herbicide must be sprayed into the air in smaller droplets than used in other types of application. Drift is less likely to occur in basal application where a more directed spray is applied at the base of the plant. The least chance for drift occurs with cut stump treatments or the use of pellets, although herbicides may evaporate slowly over time.

Commonly, damage to vegetation adjacent to rights-of-way is used as an indication of drift. However, small droplets in a strong or turbulent wind may result in concentrations of herbicide in air that are too low to visibly affect surrounding vegetation, even though measurable amounts may be moving off the right-of-way. The significance of long-range movements from the target site of low concentrations of herbicide is unclear. Because herbicides are applied for only a short time and in a small area, the concentrations of herbicides which will result on a regional level are probably insignificant. A greater potential for problems exists in areas close to the rights-of-way where levels too low to cause visible vegetation damage may be high enough to affect humans and other organisms. Diquat applications are of particular concern, since information from Chevron Chemical Company states that "breathing spray mist may cause nasal, throat, and respiratory tract irritations." The situation is less clear in regard to the other herbicides. Acute effects are generally not likely to occur; however, scientific questions remain concerning the concentrations of a chemical and frequency of exposure likely to cause long-term effects.

Surfactants

A number of comments on the draft GEIR requested additional information on inert ingredients in herbicide formulations, specifically adjuvants and surfactants. An adjuvant is any material which increases the bioactivity of the active ingredient. Surfactants, also known as surface active agents, are compounds that reduce the surface tension between two liquids or between a liquid and a solid. (The term "surfactants" actually describes two sets of compounds--one which facilitates mixing of

the active ingredient in its carrier and one which increases the contact and absorption of the active ingredient on the surface of the leaf. In the first sense, a surfactant accomplishes the same purpose as an emulsifier.)

Both surfactants and adjuvants are numerous and varied in their chemical structure. In fact, there may easily be more surfactants in number than pesticides as a class of compounds. Without knowing the chemical structure and mode of action, it is difficult to generalize about their environmental impact, except to say that surfactants are likely to increase mobility by facilitating the movement of herbicide with the flow of water. (On the other hand, if the surfactant is designed to increase absorption by target plants, it may result in less active ingredients released to the environment.) Reviewers of the draft GEIR were particularly concerned about bee toxicity; again, it is not possible to assess this without specific tests for bee toxicity. (Such tests, however, have been done for diesel oil, another concern of reviewers, and the carrier has been found to be toxic to bees.) As indicated in Appendix II, toxicity to fish has been associated with surfactants used in a glyphosate formulation. As of this writing (June 1984), Monsanto is still using this surfactant in its glyphosate formulation.

During the preparation of the draft GEIR, very little information was found on the toxicity or mobility of the various inert ingredients used in the herbicide formulations. The Commonwealth does not routinely require information on the inert ingredients of a herbicide formulation. Only when a particular herbicide is designated as being of concern will the Commonwealth decide what additional information is required from the manufacturer. If necessary, additional information required may include data on the effects of the inert ingredients in the formulation. (On a routine basis, the only data required by the Commonwealth to register a herbicide is a copy of the label, a technical data sheet, and a copy of the registration form.)

Risk Assessment

In the review of the draft GEIR, a question was raised regarding whether exposure to these herbicides will result in harm to humans or other organisms. This question must be answered on a case-by-case basis.

Given the scope of this report, it is not possible to assess whether exposure will result in a physiological response. Such a determination would require an answer to two questions: 1) what amounts of herbicide reach people by various routes of exposure, and 2) would those amounts trigger a physiological response? The first question requires a model for each route of transport, shown in Table 5. These models would have to estimate the amount of herbicide remaining after various dispersion and degradation mechanisms acted on the herbicide en route from the site of application. The second question, predicting the physiological response to the estimated exposure levels, requires another kind of modeling effort. For the various toxicity tests (chronic oral, teratogenity, subchronic inhalation, etc.), "no-effect levels" need to be determined; i.e., the highest dosage level at which no adverse effects are observed. After dividing by a safety factor (commonly 100 or 1,000), an allowable daily intake level would then need to be determined for each route of exposure. These levels must then be compared to the predicted exposure level to determine the probability that a physiological response might occur.

Data Checklist

Reviewers requested that a checklist of required information be provided so that new herbicides can be evaluated as they enter the market.

The following is a checklist of the types of information which should be considered when evaluating a pesticide. The list is taken from EPA's delineation of data, which must be submitted as part of a registration application. Not all information is required by EPA for every product or use.

Product Information

Product identity and disclosure of ingredients

Description of manufacturing process

Discussion of the formation of unintentional ingredients

Declaration and certification of ingredient limits

Product analytical methods and data

Physical/chemical properties:

- Color
- Odor
- Melting point
- Solubility
- Stability
- Octanol/water partition coefficient
- Physical state
- Density or specific gravity
- Vapor pressure
- pH
- Dissociation constant
- Flammability
- Oxidizing or reducing action
- Explosiveness
- Miscibility
- Viscosity
- Corrosion characteristics

Toxicity

Acute oral toxicity

Acute dermal toxicity

Acute inhalation toxicity

Primary dermal irritation

Dermal sensitization

90-day oral toxicity

21-day dermal toxicity

90-day dermal toxicity

90-day inhalation toxicity

Acute delayed neurotoxicity

90-day neurotoxicity

Chronic feeding test

Oncogenicity

Teratogenicity

Reproduction

Mutagenicity

Ecological Effects

Acute avian toxicity

Avian dietary toxicity

Avian reproduction

Wild mammal toxicity

Simulated and actual field tests on mammals and birds

Acute fish toxicity (warm- and cold-water fish)

Acute toxicity to aquatic invertebrates

Acute toxicity to estuarine and marine organisms

Effects on early life stages of fish

Effects on fish lifecycle

Effects on acquatic invertebrate life cycles

Accumulation in aquatic organisms

Simulated or actual field tests on aquatic organisms

Honeybee acute contact toxicity

Honeybee toxicity of residues on foliage

Wild bee toxicity of residues on foliage

Honey bee subacute feeding toxicity

Field tests on pollinators

Acute toxicity to aquatic insects

Aquatic insect lifecycle effects

Simulated or actual field tests on aquatic insects

Effects on predators and parasites

Target area phytotoxicity

Non-target area phytotoxicity:

- Seed germination/seedling emergence
- Vegetative vigor
- Aquatic plant growth
- Field studies on terrestrial and aquatic plants

Metabolism and Residues

Uptake, distribution, and metabolism in plants

Metabolism in food-producing animals

Domestic animal safety

Analytic methods for residue analysis

Residues in:

- Potable water

Fish
Processed foods
Raw agricultural commodities
Meat, milk, poultry, and eggs

Environmental Fate

Hydrolysis

Photodegradation in water, soil, and air

Volatilization

Aerobic and anaerobic soil metabolism

Aerobic and anaerobic aquatic metabolism

Effects of microbes on pesticide

Effects of pesticide on microbes

Leaching and adsorption/desorption

Field dissipation in terrestrial and aquatic systems, forests, and in long-term studies, and as combination and tank mixes

Accumulation in rotational crops (confined and field), in irrigated crops, and in fish and aquatic invertebrates

The Regulation of Pesticides in Massachusetts

Several reviewers asked for more information regarding pesticide regulation in Massachusetts. The following section was written by the Massachusetts Department of Food and Agriculture.

In order to understand the current pesticide-regulatory scheme in Massachusetts, it is necessary to take a brief look at the history of the regulation of pesticides in the United States.

History of Federal Regulation of Pesticides

The U.S. Congress passed the Federal Insecticide, Fungicide and Rodenticide Act (FIFRA) in 1947. This law gave the mandate for regulating pesticides to the U.S. Department of Agriculture (USDA). The law required federal registration of pesticides and specific labeling on each product to facilitate its identification as it was distributed from one state to the next.

The requirements for pesticide registration at that time consisted of efficacy data and some acute and chronic toxicity data.

In 1970, the Environmental Protection Agency (EPA) was formed and given broad jurisdiction to regulate the presence of chemicals in air, soil, and water. Part of these duties was to regulate pesticides, with the mandate coming from two laws: 1) the amended FIFRA (1972 and 1978) and 2) the Pesticide Amendment to the Federal Food, Drug and Cosmetic Act (FFDCA).

The amended FIFRA greatly increased the data requirements needed to support pesticide registration. It also established two categories of pesticide classification, general use, and restricted use.

General-use pesticides are generally available for purchase by anyone, and there are no restrictions for use except those specified on the label. Restricted-use pesticides can only by sold by licensed dealers, can only be purchased by certified individuals and can only be used by either a certified individual or someone under their direct supervision. (The license and certification process is explained below.)

The amended FIFRA also included a mandate to each state for the development of a state plan to enforce FIFRA and to license/certify dealers and applicators.

The Pesticide Amendment to the FFDCA established limits (tolerances) for pesticide residues in food or feed crops. The tolerances are established by the EPA and are enforceable. The monitoring of foods to ensure compliance is conducted by the Federal Food and Drug Administration (FDA) in their Market Basket Survey.

Federal Pesticide Registration Process

1. Registration. The amended FIFRA (1978) requires that registrants submit certain data to support the registration of any new pesticide products. These requirements were only recently finalized in

regulations (40 CRF Part 158, 49 FR 42881, October 24, 1984) and include data on product chemistry, residue chemistry, environmental fate, toxicology, reentry protection, spray drift, effects on wildlife and aquatic organisms, and more.

The regulations identify documents known as Pesticide Assessment Guidelines as listing the ". . . standards for conducting acceptable tests, guidance on evaluation and reporting of data, definition of terms, further guidance on when data are required, and examples of acceptable protocols." These guidelines are available through the National Technical Information Service, 5285 Port Royal Rd., Springfield, VA 22161 (703/487-4650).

2. Re-registration. The amended FIFRA (1978) requires that the EPA review all pesticide active ingredients registered on or before January 1, 1977, through the Registration Standards Program.

"The Registration Standard Program involves a thorough review of the scientific data base underlying pesticide regulations and an identification of essential but missing studies which may not have been required when the product was initially registered or studies that are now considered insufficient." (Taken from the preamble of one of the Registration Standards issued.)

Once the EPA has assessed the data supporting the registration of an active ingredient, a document is issued (a Registration Standard) which details the federal regulatory position for the registrations of all pesticides containing that active ingredient along with the rationale behind this position.

3. Special Review. This process--formerly known as RPAR (Rebuttable Presumption Against Registration)--allows the EPA to consider new information regarding a potential for adverse effect on human health or the environment of a chemical which has been registered or reregistered.

The criteria ("triggers") which must be met or exceeded in order for a pesticide to be placed on Special Review have been identified in regulations (40 CFR 162.11).

The Special Review process involves three stages and can take up to several years before a regulatory decision is made.

History of Massachusetts Regulation of Pesticides

The Massachusetts Department of Public Health (DPH) was given authority to regulate pesticides in the early 1960s through Chapter 94B of the Massachusetts General Laws. The Pesticide Board was placed in the DPH in 1962 and was given the mandate to register pesticides and control the use and application of pesticides. In 1963, the Commonwealth began a program to license and train pesticide applicators.

The Pesticide Board was moved from the DPH to the newly formed Department of Environmental Quality Engineering (DEQE) in 1975. However, the administrative duties concerning pesticide registration (mail-out of applications, receipt of fees, approving applications) remained in the Division of Food and Drug of the DPH.

In 1978, in response to the amended FIFRA mandate to states to develop a state plan to enforce the federal law and to maintain a licensing/certification program, the Massachusetts Legislature passed the Massachusetts Pesticide Control Act (MPCA, Chaper 132B of the MGL). This law placed the Pesticide Board in the Department of Food and Agriculture (DFA), with the day-to-day work carried out by the Pesticide Bureau.

Present Pesticide Regulatory Scheme in Massachusetts

Through the MPCA, there are three entities involved in the Massachusetts pesticide-regulatory scheme--the Pesticide Bureau, the Pesticide Board, and the Pesticide Board Subcommittee.

1. Pesticide Bureau. The Bureau has five main functions:

1) Licensing/Certification Program. The Bureau maintains a program to license and/or certify individuals who wish to use pesticides commercially, sell restricted-use pesticides, or purchase and use restricted-use pesticides. There are four categories of licensing/certification.

The process requires that an individual purchase the appropriate study manual from the Cooperative Extension Service, study it, and then take the exam for the license or certification category of interest. Once the exam has been passed, the individual is eligible for a license or certification.

2) Enforcement. The Bureau enforces both the FIFRA and the MPCA by conducting routine inspections and investigations of use/misuse pesticide applications.

Routine inspections include inspecting establishments which manufacture pesticides, retail outlets which sell general-use pesticides, and outlets managed by licensed dealers which sell restricted-use pesticides.

Use/misuse investigations involve answering consumer complaints, observing pesticide applications by licensed/certified individuals to ensure compliance with the label and laws, and inspecting the records of pesticide application at commercial-applicator establishments.

3) Registration. In the Fall of 1982, the administrative duties surrounding the registration of pesticides was moved from the Division of Food and Drug (DPH) to the Bureau. Therefore, the mailing out of applications, receipt and processing of fees, and the approval of re-registrations only is carried out by the Bureau. (See Pesticide Board Subcommittee.)

4) Education. As a part of a state agency, the Bureau is obligated to educate the general public on the proper use and handling of pesticides.

5) Staff to the Pesticide Board and Pesticide Board Subcommittee. The Bureau provides a variety of support functions for the Board and Subcommittee.

2. Pesticide Board. Section 3 of the MPCA establishes a Pesticide Board consisting of 13 members: six ex-officio members representing

various state agencies, and seven gubernatorial appointees representing various interest groups.

The Board meets approximately six to eight times a year. The functions of the Board are to approve of Department (Bureau) regulations and policies and to act as an appeal board for grievances incurred as a result of Bureau enforcement actions or Subcommittee registration decisions (see below).

3. Pesticide Board Subcommittee. Section 3A of the MPCA establishes a Pesticide Board Subcommittee consisting of five members, all of which are on the Pesticide Board. The five members include four ex-officio members and one gubernatorial appointee.

The function and responsibility of the Subcommittee is to register all pesticides distributed, sold, or used in Massachusetts.

Since the Subcommittee only meets between 10 and 12 times a year, it has delegated to the Bureau the authority to approve pesticide re-registration applications (those products which were registered the previous year in the Commonwealth). New pesticide product registrations are reviewed by the Subcommittee prior to approval or disapproval.

The Subcommittee is also responsible for registering Experimental Use Permits (EUPs--permits granted by the EPA to allow the use of a pesticide to generate data to support an eventual, "normal" registration application) and Special Local Needs Registrations (SLNs or 24Cs--the mechanism by which a state can register a use of a product not on the federal registration of that product to meet a Special Local Need).

It should be noted that the FIFRA allows states to be more restrictive than the EPA regarding the registration status of pesticides in that particular state.

EVALUATION OF ALTERNATIVES

The choice of the best alternative for a particular right-of-way segment depends on a number of conditions, such as

- weather conditions
- season of the year
- difficulty of the terrain
- cost-effectiveness
- potential for impact.

This section presents a discussion of the chemical and physical control alternatives with regard to these considerations. Biological control is not evaluated as an alternative in this section because, as discussed above, it is not yet a possible replacement for chemical or physical alternatives. Fire is also not included in this evaluation because, again, current research has not progressed far enough to alleviate the concerns about its use as a control measure in Massachusetts.

Flexibility

Part of the choice of the best alternative involves constraints imposed by difficult terrain, by particular seasons of the year, and by adverse weather conditions. Table 7 shows the limitations imposed by these considerations on such alternatives as hand-cutting and mowing, and various methods of herbicide application. Information on some of the more commonly used herbicides is also included.

As shown in Table 7, hand-cutting and mowing (as an example of mechanical control) are primarily limited by terrain. Hand-cutting with chain-saws becomes dangerous in areas with steep slopes, especially if they are wet. Mechanical control must have a firm, reasonably flat surface for operation, and therefore its use is limited in rocky areas, in areas with soft or wet soils, ledges and steep slopes, and in areas with stumps. Physical control methods are not limited by season or weather except by deep snow cover.

The constraints on chemical control methods are often imposed by weather or season. Foliar sprays, of course, can be applied only

TABLE 7

LIMITATIONS IMPOSED BY WEATHER, SEASON AND TERRAIN

Treatment		Conditions Under Which Treatment CANNOT Be Used*		
		Weather	Season	Terrain
Physical				
Handcutting		Deep snow cover	No limitation	Steep slopes, wet sites
Mowing		"	"	Soft, wet sites, rocks ledge, steep slopes, stumps
Chemical				
Application Method				
Foliar -	Powered Backpack	Rain, high wind	Late fall/winter/early spring	No limitations
	Hydraulic	Rain	Late fall/winter/early spring	Very steep slopes, large rocks, wet sites
Basal -	Backpack	Rain/snow/deep snow cover	No limitations	Standing water
	Hydraulic	"	"	Standing water, very steep slopes, large rocks wet sites
Cut stump treatment		"	High sap flow in late winter, early spring	No limitations

* Information provided by the New England Power Company

TABLE 7 (CONTINUED)

LIMITATIONS IMPOSED BY WEATHER, SEASON AND TERRAIN

Treatment	Conditions Under Which Treatment CANNOT Be Used		
	Weather	Season	Terrain
Individual Herbicides			
Tordon 101® (2,4-D and picloram)	Rain	Fall/winter and early spring	Steep slopes near sensitive areas, standing water
Tordon RTU®(2,4-D and picloram)	Rain/snow/deep snow cover	High sap flow in late winter, early spring	No limitations
Krenite®	Rain	Fall/winter/spring/early summer	Directly into water
Banvel 720® (dicamba and 2,4-D)	"	Fall/winter/early spring	Steep slopes near sensitive sites
Banvel 520® (dicamba and 2,4-D)	Rain/snow/deep snow cover	No limitations	Steep slopes near sensitive sites, standing water
Banvel CST® (dicamba)	"	High sap flow in late winter early spring	No limitations
Roundup® (glyphosate)	Rain	Fall/winter/spring	Standing water
Garlon-4® (triclopyr)	Rain/snow/deep snow cover	No limitations	"
Garlon-3A® (triclopyr)	Rain	Fall/winter/early spring	Standing water

during the summer or late spring or early fall and are limited by rain and wind conditions that would result in reduced coverage of the leaves and movement away from the target plant. When hydraulic sprayers are used, terrain considerations become important, as the maneuverability of the machinery is limited in wet areas and in areas of very steep slopes and large rocks. Basal spraying can be done at any time of the year except during periods when the lower part of the stem is covered by deep snow. Basal spraying is also limited by rain and snow, which could wash the material off of the bark during or after application. Cut stump treatments can also be performed at any time of the year except for a brief period in late winter or early spring when high sap flow prevents the translocation of the herbicides into the roots. Like basal treatment, treatment of cut stumps is not possible during periods of deep snow cover or when rain or snow would wash the material from the stump surface.

Table 7 shows that the limitations imposed by weather, season, and terrain vary considerably with individual herbicides. Krenite®, for instance, can be applied only late summer, during the formation of buds for next year's growth. Other herbicides are limited by their method of application. Tordon RTU® (2,4-D and picloram) is used on rights-of-way as a cut stump treatment, and thus cannot be used during periods of high sap flow as explained above.

Cost

The following tables present cost estimates of various treatments of various types of rights-of-way. They can be used as an overall indication of vegetation control costs, although individual segments of rights-of-way may differ considerably. The primary factor which accounts for this variability is the number of stems per acre, or the degree of stability of the right-of-way. An "out-of-control" right-of-way may have as much as one stem per square foot while a stable right-of-way may have only a few stems per acre. While the out-of-control right-of-way could require use of a large scale rotary mower followed by an extensive herbicide treatment, the stable right-of-way may be maintained by one person with a back pack sprayer

TABLE 8

COST ESTIMATES

Type of Right-of-Way	Type of Treatment	Cost	Specific Conditions or Treatment	Source of Information
Utility	Herbicide	$90/acre	---	New England Power and Massachusetts Electric
		$60-92/acre	See table	New England Power and Massachusetts Electric
		$55/acre	On a stable row (low stems/acre)	Boston Edison
		$35-88/acre	See table	USDA information as reported by Arthur D. Little, Inc. (1978 data)
		$50/acre	Tordon 101® Kuron® (1.01 gal./acre)	Vegetation Control Services, Inc. (1978 data)
		$60/acre	Tordon 101® (1.46 gal./acre)	Vegetation Control Services, Inc. (1978 data)
		$77/acre	Krenite® (0.8 gal./acre)	Vegetation Control Services, Inc. (1983 data)
		$57/acre	Krenite® (0.3 gal./acre)	Vegetation Control Services, Inc. (1983 data)
Utility	Mechanical	$160-250/acre	---	New England Power and Massachusetts Electric
		$170/acre	Stable right-of-way (low stems/acre)	Boston Edison

TABLE 8 (CONTINUED)

COST ESTIMATES

Type of Right-of-Way	Type of Treatment	Cost	Specific Conditions or Treatment	Source of Information
Utility	Mechanical (continued)	2.5 times cost of herbicide treatment for any given row	---	Northeast Utilities
		$183/acre	---	National Rural Electric Cooperative Assoc. Survey (1977)
		$150-200/acre	---	Arthur D. Little report (1979)
Utility	Manual Cutting	$500/acre	---	New England Power and Massachusetts Electric
		$125/acre	Stable right-of-way (low stems/acre)	Boston Edison
		$100-400/acre	---	Citizens for Environmental Protection, Charleston, W. Virginia (landowner demonstration project)
		4.8 times cost of herbicide treatment for any given row	---	Northeast Utilities
		$657/acre	---	National Rural Electric Cooperative Assoc. Survey (1977)

TABLE 8 (CONTINUED)

COST ESTIMATES

Type of Right-of-Way	Type of Treatment	Cost	Specific Conditions or Treatment	Source of Information
Utility	Manual cutting	$800-1000/acre	Extreme or out-of-control right-of-way (high stems/acre)	Arthur D. Little Inc. report (1979)
Railroad	Herbicide on ballast	$160/mile	---	Providence & Worcester Railroad Co.
		$78/mile	---	Providence & Worcester Railroad Co.
	Brush control-herbicide	$200-300/mile	---	Providence & Worcester Railroad Co.
		$60-160/mile	---	Massachusetts Railway Assoc.
	Brush control-brush cutter	$1750/mile	---	Providence & Worcester Railroad Co.
		$800/mile	---	Providence & Worcester Railroad Co.
		$200-6500/mile	---	Massachusetts Railway Assoc.
	Brush control-manual	$1300/mile	---	Massachusetts Railway Assoc.

TABLE 9

HERBICIDE COST PER ACRE BY TYPE OF APPLICATION*

Type of Treatment	Herbicides	Average Cost per 100 Gal.	Quantity of Spray per Acre	Average Cost per Acre
Foliar	2,4-D dicamba Tordon 101® (2,4-D plus picloram)	$26.03	200 gal. (diluted)	$40.06
Basal	2,4-D dicamba	$56.31	80 gal.	$85.05(a)
Stump spray after cutting	2,4-D	$56.31	45 gal.	$47.84(b)

(a) Includes cost of 80 gallons of oil at $0.50 per gallon.

(b) Includes cost of 45 gallons of oil at $0.50 per gallon.

* USDA data reported by Arthur D. Little, Inc. (1979)

TABLE 10

SPECIFIC RIGHTS-OF-WAY OF NEW ENGLAND POWER COMPANY:
COST PER ACRE OF VARIOUS TREATMENTS

Area	Method/ Material	Acres	Cost	Cost/Acre
Fitchburg/ Ashburnham	Krenite®	319 A	$25,100	$78.68
Warwick/Gardner	Krenite®	245 A	$19,200	$78.37
Athol	Krenite®	90 A	$ 8,300	$92.22
Erving/ Petersham	Krenite®	375 A	$20,924	$55.80
Vernon, Vt./ Warwick	Krenite®	78 A	$ 6,050	$77.56
Brattleboro/ Bellows Falls	Garlon 3A®	243 A	$14,692	$60.46
No. Reading	Handcutting	10.9 A	$ 5,492	$503.85
Mullbury	Tractor mowing	11.4 A	$ 3,146	$276.00
Oxford	Tractor mowing	10.5 A	$ 1,669	$159.00
Charlton	Tractor mowing	10.6 A	$ 890	$84.00

(Herbicide cost includes some handcuttings at streams, gardens, etc.)

or chain saw walking the right-of-way, eliminating individual trees. Another important factor in cost is the amount of clean-up required, i.e., whether chipping or removal of brush is included in the cost estimate. Labor intensive clean-up efforts can easily double the cost of the control treatment.

Some reviewers of the draft GEIR questioned the reliability of the sources of cost data included in the draft since they were provided by those who have a vested interest in showing that non-herbicide controls are unreasonably expensive, i.e., utilities and railroad companies. These data sources were used because in general they are the only ones who are generating and recording cost data. One interesting exception is the data from Citizens for Environmental Protection from Charleston, West Virginia. Manual control costs from their landowner demonstration project ranged from $100 to $400/acre compared to $125 to $657/acre estimated by utilities and their associations.

Environmental Impact

The impacts of the herbicides covered in this report are addressed in the literature review presented in Appendix II and in the summaries of that literature provided in a previous section. The environmental impacts of physical control methods (excluding fire) include a number of minor impacts such as increased noise and air pollution from the machines, and one major impact--the likelihood of causing accidents to workers. Further comparison of these different types of impact is presented below in regard to policy evaluation.

RECOMMENDATIONS

The information compiled in this report was used to develop policy recommendations for the use of herbicides on rights-of-way in Massachusetts. This policy was developed by a task force that has been assembled for this purpose. The task force, representing environmental groups, utility and railroad companies, applicators, local officials, and various state agencies, among others, generated the following document.

Introduction

Lately, despite the fact that herbicides are used for many other purposes as well, public concern has focused on the use of herbicides to maintain utility and railroad rights-of-way. The use of herbicides on rights-of-way to control vegetation accounts for approximately 17 to 29 percent of the total use of herbicides in Massachusetts.

In order to estimate the potential environmental impact of herbicides to maintain utility and railroad rights-of-way in Massachusetts, the Executive Office of Environmental Affairs commissioned the preparation of a Generic Environmental Impact Report on this subject. Preparation of the Report has been overseen by the MEPA Unit of EOEA with financial support provided by the Department of Food and Agriculture. The MEPA Unit also assembled a Task Force representative of the full range of interests concerned with herbicide use to review the Impact Report and to recommend policies the state should pursue with respect to the control of herbicide use.

The Task Force has considered four general questions in its effort to translate the findings of the Impact Report into policy recommendations. These are:

1. Should the state program for regulating the use of herbicides on rights-of-way be upgraded?

2. Should the state establish procedures that will streamline and coordinate the regulation of herbicide use?

3. Should the state promote the use of "integrated management techniques" for the control of vegetation along rights-of-way?

4. Should the state classify or group herbicides according to common characteristics and regulate them accordingly?

In each case, the Task Force considered how such recommendations would be implemented if the answer were yes. The Task Force answered the first three questions with a resounding yes. The fourth question could not be satisfactorily answered at this time. The recommendations and how they would be implemented are discussed below. The recommendations should be understood as a whole and implemented in that fashion. Selective implementation would eliminate the spirit and elements of compromise which have led to its endorsement by the Task Force.

The six recommendations are:

1. THE EXISTING STATE PROGRAM WHICH REGULATES THE APPLICATION OF HERBICIDES TO RIGHTS-OF-WAY SHOULD BE SIGNIFICANTLY ENHANCED.

2. THE STATE SHOULD REQUIRE THE USE OF INTEGRATED MANAGEMENT TECHNIQUES FOR RIGHT-OF-WAY MAINTENANCE BY REQUIRING COMPLIANCE WITH APPROVED VEGETATION MANAGEMENT PLANS BY ALL RIGHT-OF-WAY OWNERS.

3. THE STATE REGULATIONS SHOULD DEFINE GEOGRAPHIC AREAS OF SPECIAL SENSITIVITY TO HERBICIDE APPLICATIONS AND RESTRICT SUCH APPLICATION IN THESE AREAS.

4. THE COMMONWEALTH OF MASSACHUSETTS SHOULD COORDINATE ITS EFFORTS WITH THOSE OF LOCAL GOVERNMENTS TO ESTABLISH PROCEDURES THAT WILL STREAMLINE THE REGULATION OF

HERBICIDE APPLICATIONS. THE SUBSTANTIVE AND PROCEDURAL REQUIREMENTS FOR OBTAINING AN APPROVED VEGETATION MANAGEMENT PLAN SHOULD INTEGRATE THE INTERESTS OF THE WETLANDS PROTECTION ACT AS THEY PERTAIN TO VEGETATION MANAGEMENT ON RIGHTS-OF-WAY.

5. THE STATE SHOULD ESTABLISH PROCEDURES WHICH GUARANTEE AMPLE OPPORTUNITY FOR PUBLIC REVIEW AND COMMENT ON RIGHT-OF-WAY MAINTENANCE PLANS AND THE REGULATIONS WHICH GOVERN THEM.

6. REVIEW OF THE CHEMICAL AND OTHER PROPERTIES OF PESTICIDES SHOULD TAKE PLACE IN THE PESTICIDE REGISTRATION PROCESS AND IN CONSIDERATION OF VEGETATION MANAGEMENT PLANS. CLASSIFICATION OF HERBICIDES ACCORDING TO CERTAIN OF THEIR SIMILARITIES SHOULD BE GIVEN FURTHER CONSIDERATION BY STATE REGULATORS. HOWEVER, PROMULGATION OF NEW REGULATIONS FOR CONTROLLING HERBICIDE APPLICATIONS SHOULD NOT BE DELAYED NOR DEPEND UPON SUCH CLASSIFICATION.

Attachment One lists the members of the Task Force.

Recommendation 1

THE EXISTING STATE PROGRAM WHICH REGULATES THE APPLICATION OF HERBICIDES TO RIGHTS-OF-WAY SHOULD BE SIGNIFICANTLY ENHANCED.

Discussion

An enhanced state-level program regulating methods used to control vegetation along utility and railroad rights-of-way is urgently needed. The quality of current right-of-way maintenance practices varies, even within existing regulations and guidelines. An effective and reliable

state-level program will alleviate the need perceived now by some for aggressive efforts to regulate these practices at the local government level. Citizens, local officials, state officials, utilities, railroads, herbicide applicators, and environmental groups will all most certainly benefit, albeit in different ways, from a strong, comprehensive state regulatory program.

The goal of an enhanced, state-level regulatory program should be to eliminate threats to public health and the environment that might be caused by herbicide application and, wherever possible, encourage the use of alternatives to herbicide use. The public's interest in adequate protection of public health should be accorded consideration equal to the consideration given to adequate right-of-way maintenance in public policy decisions. In practical terms, the program should keep the application of herbicides to a minimum.

The success of the regulatory program recommended here depends on the submission of detailed reports on planned activities by utilities and railroads which must be reviewed, approved, and monitored by technically qualified personnel at the state level. This workload will require allocation of additional staff to the state agency(ies) reponsible for carrying out the program. Given the difficulties of securing additional staff through the state budget process, attention should be given to mounting a unified broad-based campaign to secure support for funding these positions. All interests will be served by capable implementation of this program. All will be harmed if this is not achieved.

Implementation Steps

1. The goals of the regulatory program should be embodied in statutory and regulatory language so that the authority to promulgate and enforce the program is unambiguous and widely recognized.

2. The Department of Food and Agriculture, with the approval of the Pesticide Board, should promulgate new regulations that keep the

application of herbicides to a minimum and encourage the use of "integrated management techniques". (See Recommendation 2 for the details of this program.)

3. The Legislature should increase appropriations for at least eight (8) new positions in the Department of Food and Agriculture to enable the department to implement the programs called for in these recommendations. These new positions would provide:

    a) capability in environmental analysis to carry out reviews and approvals of Vegetation Management Plans (2 positions);

    b) enforcement capability to insure compliance by right-of-way owners with Vegetation Management Plans (3 positions: 2 inspectors and 1 attorney);

    c) capability in public information programs to coordinate public reviews of Vegetation Management Plans (1 position); and

    d) capability in analysis of toxicology and environmental fate to carry out pesticide product assessments for the Pesticide Board (2 positions).

4. The Legislature should increase appropriations for at least three new positions in the Department of Environmental Quality Engineering to enable the Department to implement the programs called for in these recommendations. These new positions would provide:

    a) for increased hydrogeological capability to identify and evaluate the extent of areas which contribute water to public water supplies (1 position);

    b) for increased toxicological capability to analyze the characteristics of herbicides (1 position); and

c) for increased staff in the Division of Wetlands to review and approve Vegetation Management Plans for compliance with the regulations adopted pursuant to the requirements of the Wetlands Protection Act (1 position). (See Recommendation 4, Steps 3 and 4.)

5. As soon as possible, the Pesticide Board should conduct an in-depth review of current procedures for registration of pesticides in Massachusetts. This review should clarify the relationship between the registration procedures of the U.S. Environmental Protection Agency as well as the current registration practices of the Board, identify chemicals which pose unacceptable public health risks, and, wherever possible, improve current registration practices.

Recommendation 2

THE STATE SHOULD REQUIRE THE USE OF INTEGRATED MANAGEMENT TECHNIQUES FOR RIGHT-OF-WAY MAINTENANCE BY REQUIRING COMPLIANCE WITH APPROVED VEGETATION MANAGEMENT PLANS BY ALL RIGHT-OF-WAY OWNERS.

Discussion

The quality of current right-of-way maintenance practices varies, even within existing laws and regulations. Therefore the state should require that all utilities and railroads who maintain rights-of-way submit Vegetation Management Plans. The Plans should cover the entire right-of-way system owned and operated by the utility or railroad within Massachusetts, document how the goals and regulations of the state program will be met, and should be kept current. The Plans should provide justification for any proposed herbicide use. The Plans should demonstrate that integrated management practices (i.e. the use of non-herbicide control practices wherever possible) are being practiced. No application of herbicides should be allowed in the absence of a state-approved Vegetation Management Plan. The requirements of the plan should be enforceable under provisions of the Massachusetts Pesticides Control Act.

The obligation to obtain an approved Vegetation Management Plan should be applicable to all rights-of-way operators, including state agencies and authorities.

Implementation Steps

1. Vegetation Management Plans should include four parts:

   A) Vegetation Management Master Plans
   The Master Plan should be filed by each utility and railroad with the Pesticide Board and DEQE and should describe the

overall approach each one will use to control vegetation along their right-of-way, including

1) The reasons for managing vegetation.

2) The goal of the plan (e.g. elimination of tree species, encouragement of low growing plants, vegetation free areas, etc.).

3) The methods of vegetation management proposed and conditions under which each method would be used. Type of equipment used for each method.
   a) Hand cutting
   b) Mechanical cutting
   c) Herbicide treatment by type:
      - Basal
      - Cut surface
      - Foliar
      - Soil

4) Discussion of the rationale for selection of one management method over another.

5) Characteristics of herbicides to be used.

6) Methods for control of herbicide drift.

7) Special treatment strategies for sensitive areas (See Recommendation 3.)

8) Summary discussion of environmental impacts of management plan.

9) Average treatment cycle.

10) Persons, and their qualifications, who will develop and administer the plan.

2. The state should examine the application of herbicides to rights-of-way not owned or maintained by utilities and railroads (most notably, those maintained by highway departments) and determine the extent to which the requirements of this regulatory program should be applied to all right-of-way owners and operators.

Recommendation 3

THE STATE REGULATIONS SHOULD DEFINE GEOGRAPHIC AREAS OF SPECIAL SENSITIVITY TO HERBICIDE APPLICATIONS AND RESTRICT SUCH APPLICATIONS IN THESE AREAS.

Discussion

The regulations should prescribe that these areas be treated in special ways to minimize any potential harm to public health or the environment which could be caused by inappropriate herbicide applications. Some of these areas may be best restricted from any herbicide applications at all. Some may be restricted to specific methods of application and specific herbicides.

Implementation Steps

1. The sensitive areas subject to special restrictions ought to include, though not be limited to, the following:

    a) zones of contribution to public water supplies as defined by the DEQE;
    b) private wells and water supplies;
    c) shorelines and tributaries of surface public water supplies;
    d) areas identified by DEQE as potential future water supplies;
    e) open water (lakes and streams);
    f) gardens and broad-leafed crops in the growing season;

g) other crops in growing season, and broad-leafed crops in dormant season;
h) residential areas, designated parks and recreational areas, and public gathering places;
i) schools, hospitals, and other structures used by sensitive populations;
j) wetlands;
k) critical biological areas (e.g. habitats for rare and endangered species);
l) estuaries;
m) wildlife management areas;
n) and other areas designated by the Pesticides Control Board.

2. In addition to defining these sensitive areas, the regulations should establish distances within some or all of these sensitive areas where general applications may not take place. The remainder of these sensitive areas should be subject to special precautions established by the Department of Food and Agriculture.

3. Procedures should be established in the regulations to allow for expansion or contraction of the distances from the center of sensitive areas based upon a petition showing special circumstances or based upon the development of new scientific information.

4. Within those distances where no herbicide use is otherwise allowed, some restricted use of herbicides may be allowed if, and only if, the owner has demonstrated to the satisfaction of the Department of Food and Agriculture that no alternative means of control is available and that there is an overriding public hazard resulting from the lack of herbicide application. However, this provision should not be applicable to areas subject to the Wetlands Protection Act as determined by the DEQE based upon review of Vegetation Management Plans.

Recommendation 4

THE COMMONWEALTH OF MASSACHUSETTS SHOULD COORDINATE ITS EFFORTS WITH THOSE OF LOCAL GOVERNMENTS TO ESTABLISH PROCEDURES THAT WILL STREAMLINE THE REGULATION OF HERBICIDE APPLICATIONS. THE SUBSTANTIVE AND PROCEDURAL REQUIREMENTS FOR OBTAINING AN APPROVED VEGETATION MANAGEMENT PLAN SHOULD INTEGRATE THE INTERESTS OF THE WETLANDS PROTECTION ACT AS THEY PERTAIN TO VEGETATION MANAGEMENT ON RIGHTS-OF-WAY.

Discussion

A cooperative relationship between state agencies and local governments should be reflected in the design of an enhanced, state regulatory program. The state agencies responsible for approval of utility and railroad plans for control of vegetation along rights-of-way must insure appropriate opportunity for public comment and review of proposed plans, during which time local regulatory and other concerns should be focused. The objective of these coordinated efforts should be to avoid duplicative regulation.

The state should encourage cooperative agreements between utilities or railroads and municipalities and landowners along rights-of-way to manage vegetation through the use of non-chemical alternatives to herbicides. The acceptability of these agreements depends in each case on the resolution of issues dealing with third-party liabilities and landowner consent, difficult but not unsurmountable problems. It may not be advisable to implement such agreements for the most heavily traveled railroad lines.

Implementation Steps

1. The state agencies should integrate the interests and requirements of all state-level regulatory programs dealing with herbicide application on rights-of-way in one approval process.

2. The regulations which provide for approval of Vegetation Management Plans by the Department of Food and Agriculture should be conditioned on review and approval by the Department of Environmental Quality Engineering (DEQE) of those portions of the Plans that deal with wetlands. The DEQE should be required to certify to the DFA that these portions of the Plans will result in compliance with the substantive and procedural provisions which protect the interests of the Wetlands Protection Act. If the regulations are so drawn, activities under a Plan approved by DEQE would not constitute an alteration of wetlands as defined under the Wetland Protection Act regulations.

3. If possible, the DEQE should establish and publicize criteria for evaluation of herbicide applications in wetlands which foster consistency in the review and approval of Vegetation Management Plans. Such criteria would assist local Conservation Commissions in cases where they were reviewing a Request for Determination of Applicability of the Wetlands Protection Act with regard to activities called for in a Vegetation Management Plan.

4. The state should allow municipal governments and/or landowners along rights-of-way to submit proposed alternative, non-chemical vegetation management plans to utilities and railroads if they prefer to undertake right-of-way maintenance programs of their own. These proposals should explain why the proponents believe their management program is needed. The owner/operator of the right-of-way should have the burden of showing why such plans are unacceptable. The Pesticide Board should attempt to resolve disagreements over the acceptability of an alternative management proposal in the context of its review of Vegetation Management Plans.

5. The regulations should encourage the appointment of pesticide application coordinators at the municipal level and spell out the responsibilities of such persons. These responsibilities should include keeping interested citizens aware of proposed and approved Vegetation Management Plans.

Recommendation 5

THE STATE SHOULD ESTABLISH PROCEDURES WHICH GUARANTEE AMPLE OPPORTUNITY FOR PUBLIC REVIEW AND COMMENT ON RIGHT-OF-WAY MAINTENANCE PLANS AND THE REGULATIONS WHICH GOVERN THEM.

Discussion

Recently, citizens of the Commonwealth acting on their own and through their local governments have expressed considerable concern about the application of herbicides to rights-of-way. This concern may not abate simply with the advent of a strong and comprehensive regulatory program. The state must be sure to provide all appropriate opportunities for public review and comment as new regulations are developed and implemented to be sure these concerns continue to be heard and responded to.

This is especially true with respect to the portion of the regulations dealing with sensitive areas. The proper designation of the setbacks from these areas, the way they are defined in general, and their actual location along particular rights-of-way will be critical to the successful functioning of the regulatory program. The knowledge and experience of citizens and local officials will be a valuable addition to efforts to arrive at proper designations. In particular, the regulatory program calls for determinations on the acceptability of management practices for wetlands located in rights-of-way to be elevated from the local level of Conservations Commissions to the state level as part of the review of Vegetation Management Plans. This means that local officials must be assured of notification and opportunity for comment in the review process for Vegetation Management Plans if the substantial local interests in wetlands protection available under the Act's procedures are to be maintained.

Implementation Steps

1. Both the regulations and, later, overall Vegetation Management Plans should be subjected to public review and comment before being finalized and approved. Copies of the overall plans and annual notifications should be sent to all concerned State agencies and municipalities.

2. The Department of Food and Agriculture should, at its discretion, upon request of a state, federal, or local government agency, hold public information meetings to take comments on proposed Plans. Before approving a Plan the DFA should demonstrate that concerns of commenting parties have been addressed wherever possible.

3. The regulations should establish a procedure for appeal by an aggrieved party of decisions on Vegetation Management Plans.

Recommendation 6

REVIEW OF THE CHEMICAL AND OTHER PROPERTIES OF PESTICIDES SHOULD TAKE PLACE IN THE PESTICIDE REGISTRATION PROCESS AND IN CONSIDERATION OF VEGETATION MANAGEMENT PLANS. CLASSIFICATION OF HERBICIDES ACCORDING TO CERTAIN OF THEIR SIMILARITIES SHOULD BE GIVEN FURTHER CONSIDERATION BY STATE REGULATORS. HOWEVER, PROMULGATION OF NEW REGULATIONS FOR CONTROLLING HERBICIDE APPLICATIONS SHOULD NOT BE DELAYED NOR DEPEND UPON SUCH CLASSIFICATION.

Discussion

The Task Force has found that the analysis of herbicides to determine their potential environmental and public health impact is an exceedingly

complex process. It requires careful consideration of:

- toxicity of the herbicide, (estimated according to available toxicological studies);
- evaluation of the reliability of available scientific data;
- mobility of the herbicide in varying soil types (high organic content soils yield low mobilities);
- sensitivity of the area being treated; and
- the method of herbicide treatment.

The Task Force gave considerable attention to the possible advantages and disadvantages of classifying herbicides with similar chemical or other properties. Some felt such a scheme would allow local officials and others to understand and comment on Vegetation Management Plans and the decisions of state regulators on these plans. Others felt such schemes naturally lead to over-simplified judgements and inappropriate conclusions because so many important variables cannot be included in the scheme. In general, the feeling was that educational advantages would very likely be outweighed by misunderstandings or abuse of such a scheme.

However, if a method of classification could be developed which was based on recognized scientific data and analytical methods, it could help in the review of Vegetation Management Plans. Further consideration of herbicide classification by the Pesticide Board appears warranted.

Implementation Steps

1. Current registration procedures for testing and/or evaluation of herbicides should be reviewed and, wherever possible, strengthened. (See Recommendation 1, Step 5.)

2. Vegetation Management Plans should include a full discussion of the characteristics of herbicides to be used, including summaries of relevant and available data on environmental fate and toxicology. (See Recommendation 2, Step 1.)

Attachment One
MEPA Herbicides Task Force

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Bill Febiger	Energy Facilities Siting Council
Christy Foote-Smith	MA Association of Conservation Commissions
Carol Greenleaf	Office of State Senator Amick
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APPENDIX I: SUPPLEMENTAL INFORMATION

CHAPTER 1. LEGAL FRAMEWORK*

This chapter identifies and summarizes applicable federal and state law affecting the use of herbicides for railroad and electric utility right-of-way vegetation control in Massachusetts. No attempt is made to render legal opinions, to resolve apparent conflicts in the law, or to address issues of policy. Due to space limitations, this chapter should be viewed as an overview, rather than an exhaustive treatment of the subject.

Potentially applicable statutes, regulations, and legal doctrines discussed below include: the Federal Insecticide, Fungicide and Rodenticide Act and regulations; the Massachusetts Pesticide Control Act and regulations; the Massachusetts herbicide notification statute; the Pesticide Board's interim guidelines for right-of-way applications; the Massachusetts Wetlands Protection Act and regulations; the Massachusetts Clean Water Act; several Massachusetts electric utility and railroad regulatory statutes; the Federal Railroad Safety Act and regulations; the Massachusetts Environmental Policy Act; several statutes establishing the regulatory powers of cities and towns with respect to public health, electric utilities, water supply, zoning and general bylaws; the Massachusetts Home Rule Amendment; and the doctrine of preemption. Unresolved legal issues include the scope of the existing statutory authority of state agencies to regulate herbicide use by railroads and electric utilities; and whether (and if so to what extent) local regulation of such herbicide application is preempted by state or federal law.

Description of Applicable Law

A. Pesticide Regulation

1. FIFRA

The Federal Insecticide, Fungicide and Rodenticide Act, as amended by the Federal Environmental Pesticide Act of 1972 and the Federal Pesticide Act of 1978, 7 U.S.C. §§135-136y (collectively, "FIFRA"), establishes a establishes a comprehensive federal scheme for the regulation

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of pesticides, including herbicides. FIFRA is administered by the United States Environmental Protection Agency ("EPA"). Among other things, FIFRA requires, inter alia, the registration and classification for general or restricted use of all pesticides sold in the United States (7 U.S.C. §136a), regulates the labeling of pesticides (7 U.S.C. §136a), forbids the use of a pesticide in a manner inconsistent with its labeling (7 U.S.C. §136(2)(G)), and requires that restricted-use pesticides be applied only by or under the supervision of certified applicators (7 U.S.C. §136b). FIFRA also establishes a framework within which EPA may publicly disclose health safety and environmental data submitted in support of a pesticide registration.

EPA has promulgated detailed regulations implementing FIFRA, 40 CFR §§162-180. In particular, the FIFRA regulations specify the required contents of pesticide labels, including active ingredients, warnings as to toxicological and environmental hazards, and applicable use restrictions. 40 CFR §162.10. The regulations also specify criteria for the determination by EPA as to whether a pesticide will cause "unreasonable adverse effects on the environment," in which case its registration may be denied or cancelled. 7 U.S.C. §136a-(c)(5)-(6); 40 CFR §162.11. Among the herbicides, EPA has classified only picloram as "restricted use" on the basis of its hazard to non-target vegetation. 40 CFR §162.31.

With respect to the trade secret disclosure, the recent United States Supreme Court decision of Ruckelshaus v. Monsanto Co., 52 U.S.L.W. 4886 (June 26, 1984), upheld a provision of FIFRA which relates to public disclosure of, among other things, data that has been designated by an applicant for registration as "trade secrets or commercial or financial information" under another FIFRA section, 7 U.S.C. §136h(b). The provision had been challenged by a pesticide manufacturer who argued that the disclosure of trade secrets submitted during the application process constitutes a taking of property in violation of the Fifth Amendment to the United States Constitution. The Supreme Court reasoned that the manufacturer had notice of FIFRA's disclosure provisions when it chose to submit data, except for data submitted between

1972 and 1978 under a previous version of FIFRA guaranteeing confidentiality, and that even as to 1972-1978 data, just compensation could be obtained from the federal court of claims.

Section 136h(d) enacted in 1978 and enforced by both civil and criminal penalties under Section 1361, provides as follows:

> (d) Limitations -
>
> (1) All information concerning the objectives, methodology, results, or significance of any test or experiment performed on or with a registered or previously registered pesticide ... and any information concerning the effects of such pesticide on any organism or the behavior of such pesticide in the environment . . . shall be available for disclosure to the public: . . . Provided further, That this paragraph does not authorize the disclosure of any information that -
>
> (A) discloses manufacturing or quality control processes,
>
> (B) discloses the details of any methods for testing, detecting, or measuring the quality of any deliberately added inert ingredient of a pesticide, or
>
> (C) discloses the identity or percentage quantity of any deliberately added inert ingredient of a pesticide,
>
> unless the Administrator has first determined that disclosure is necessary to protect against any unreasonable risk of injury to health or the environment.
>
> (2) Information concerning production, distribution, sale, or inventories of a pesticide that is otherwise entitled to confidential treatment under subsection (b) of this section [data designated as trade secrets or commercial or financial information] may be publicly disclosed in connection with a public proceeding to determine whether a pesticide, or any ingredient of a pesticide, causes unreasonable adverse effects on health or the environment, if the Administrator determines that such disclosure is necessary in the public interest.
>
> (3) If the Administrator proposes to disclose information described in clause (A), (B), or (C) of paragraph (1) or in paragraph (2) of the subsection, the Administrator shall notify by certified mail the submitter of such information of the intent to release such

> information. . . . During such period the data submitter may institute an action in an appropriate district court to enjoin or limit the proposed disclosure. . . . The court may enjoin disclosure, or limit the disclosure or the parties to whom disclosure shall be made. . . .

Under this provision, then, states and qualified members of the public may gain access to some information offered in support of a FIFRA registration. Other information will be made available to them if the Administrator of the EPA determines that health or environmental concerns warrant such disclosure.

State Regulation and Federal Preemption

There is a question as to whether FIFRA, as a federal act, preempts state pesticide legislation not explicitly authorized by FIFRA (and if so, to what extent). The only statutory language pertaining to state registration of pesticides is contained in section 136v(c)(1) which authorizes state registration for additional uses of federally registered pesticides to meet "special local needs". At the same time, FIFRA expressly contemplates some state regulation of federally registered pesticides, 7 U.S.C. §136v, and authorizes EPA to delegate to the states primary enforcement responsibility for pesticide use violations, 7 U.S.C. §136w-1. Courts have reached different conclusions as to the breadth of the regulatory authority embodied in section 136v(a) of FIFRA which provides that states may "regulate the sale or use of any federally registered pesticide or device in the State, but only if and to the extent the regulation does not permit any sale or use prohibited by this subchapter." Compare National Agricultural Chemical Ass'n v. Romiger, 500 F. Supp. 465 (E.D. Cal. 1980) (FIFRA does not preempt state's right to require additional data from pesticide manufacturers and distributors as condition of registration) with Pacific Construction Co. v. Branch, 428 F. Supp. 727 (D. Guam 1976) (FIFRA preempts state's authority to promulgate import restrictions).

It should be noted, however, that even if states do regulate federal registered pesticides more strictly than EPA, FIFRA prohibits any state

from "imposing any requirements for labeling or packaging in addition to or different from those required [by FIFRA]", 7 U.S.C. §136v. Thus, any state enacting more stringent use restrictions than EPA faces significant problems in communicating those restrictions.

The Massachusetts Pesticide Control Act ("MPCA") authorizes the Subcommittee of the Pesticide Board to register for use in the Commonwealth pesticides, "including pesticides that are federally registered." G.L. c.132B § 7. MPCA further provides that the Subcommittee "may require of applicants for pesticide registrations any information that it deems necessary to determine whether, or how, the pesticide should be registered." Id. Regulations promulgated pursuant to MPCA state that "[t]he Subcommittee may register or refuse to register any pesticide for distribution, sale or use in the Commonwealth" according to the standards and procedures set forth in 333 C.M.R. section 8.00. Section 8.05. On their face, these statutory provisions and regulations appear to give the Commonwealth broad authority to establish state registration standards and procedures beyond those which may be authorized by FIFRA.

A number of legal issues might be raised, however, if the Commonwealth of Massachusetts were, for example, to revise its pesticide registration program to require submission of health, safety and environmental data as a condition of state registration or re-registration. A significant factor motivating such concerns is the absence of any trade secret protection in the MPCA. Without such protection, any data submitted by an applicant would be subject to the Massachusetts Public Records Act, M.G.L. c. 66 § 10, and would have to be made available to the public upon request. Under Monsanto, the possibility of such broad disclosure might require applicants to make business judgements weighing the benefits of registration in Massachusetts against the costs of their divulging trade secrets.

Among the legal issues that would be presented by a more stringent state registration program are the following:

(1) Would such a program be preempted under the Supremacy Clause by the existing FIFRA registration process?

(2) Would such a program result in an unconstitutional taking of property? See Monsanto. Is the Fifth Amendment Taking Clause applicable to a state as opposed to the federal government?

(3) Would such a program be an unconstitutional violation of due process rights?

(4) Would such a program violate the Commerce Clause of Article I of the federal Constitution by unlawfully restraining interstate commerce?

(5) Even if not unconstitutional, would such a program violate federal or state trade secret statutes?

(6) Even if not unconstitutional, would such a program jeopardize common law trade secret protection?

The resolution of these issues could depend on the scope and details of such an expanded state registration program.

2. The Massachusetts Pesticide Control Act

The Massachusetts Pesticide Control Act, M.G.L. c. 132B ("MPCA"), enacted in 1978, establishes a comprehensive state pesticide regulatory program closely patterned after the federal program under FIFRA. The MPCA established the Massachusetts Pesticide Board ("the Board") within the Department of Food and Agriculture, which implements the Massachusetts pesticide program. G.L. c. 132B, §3. The MPCA provides for state registration of pesticides (id., §7), forbids the distribution of pesticides not registered with the Board (id., §6), forbids the use of pesticides inconsistent with their labeling or use restrictions (id., §6A), prohibits the use of restricted-use pesticides except by or under the supervision of certified applicators (id., §6A), and provides for state certification of applicators (id., §10).

The MPCA is implemented by regulations promulgated by the Board, 333 CMR §2.00, et seq. These regulations provide that applicators shall use pesticides so as to prevent "unreasonable adverse health effects on

the non-target environment," that right-of-way applications shall be conducted "to minimize the extent and duration of foliar brown-out," that pesticide applications near or adjacent to public water supplies "shall be made in such a manner as to minimize the risk of adverse effects to such water supplies," and that for applications of restricted or state-limited use pesticides to areas of more than 25 acres, permission must be received from the Board and notice given to the appropriate local official. 333 CMR §10.03(19)-(21). The MPCA regulations also provide detailed standards for the certification of applicators for particular categories of uses (e.g., "right-of-way pest control"). 333 CMR §10.05. Violators of the MPCA or Pesticide Regulations are subject to civil or criminal penalties. G.L. c. 132B, §14; 333 CMR §10.17. On July 1, 1980, EPA delegated primary enforcement authority of FIFRA in Massachusetts to the Board through a federal-state cooperative agreement.

3. The Notification Statute

Chapter 722 of the Acts of 1981, G.L. c. 132B, §6B, requires that any electric or other "utility company" (which the Board interprets to include railroads), prior to any application of herbicides to their rights-of-way, notify the mayor, city manager or board of selectmen, and the conservation commission, of the town in which the application is to be done, by registered mail 21 days in advance of the spraying, that herbicide spraying will be done. The notice is to include the approximate dates of the application, the type of herbicide, information supplied by the manufacturer (e.g., the label), and identification of the contractor or utility employee responsible for the application. Herbicide application must be done within 10 days of the dates included in the notice. The notification statute is silent on the subject of local regulation of such herbicide use.

4. Pesticide Board Interim Guidelines

In 1982, the Board promulgated two sets of "interim guidelines" concerning herbicide applications to railroad and utility rights-of-way. There are the "interim Guidelines Relative to the Use of Herbicides on Ballast Area of Railroad Layouts in Massachusetts" (revised October 15, 1982),

and the "Interim Guidelines Relative to the Use of Herbicides to Control Woody Vegetation on Railroad Layouts and Right-of-Ways in Massachusetts" (October 15, 1980). The latter is applicable to electric utility rights-of-way as well as railroads. Both sets of guidelines are intended to protect drinking water supplies from herbicide contamination, and prohibit herbicide application within prescribed distances of public and private wells, surface water supplies, and tributaries thereof. Both sets of guidelines also contain "general use guidelines" to minimize herbicide drift or runoff. The Board intends to promulgate definitive regulations to replace the interim guidelines on the basis of this statewide Generic Environmental Impact Report on the control of vegetation on utility rights-of-way and railroad layouts, if the Board determines that such regulations are necessary.

B. Wetlands Regulation

1. General Regulatory Scheme

The Massachusetts Wetlands Protection Act, G.L. c. 131, §40 (the "Act") imposes pre-construction review upon projects affecting wetlands. The Act prohibits the removal, filling, dredging, or alteration of certain statutorily defined wetland resource areas ("wetlands")[1] without first filing a Notice of Intent with the local conservation commission and obtaining from the commission a permit known as an "order of conditions" regulating the proposed work, so as to protect the affected wetlands values. Regulatory jurisdiction under the Act attaches to any activity proposed or undertaken within wetlands subject to protection under the Act, or within 100 feet of certain such areas

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[1] Wetland resource areas protected by the Act include "any bank, fresh water wetland, coastal wetland, beach dune, flat, marsh, meadow, or swamp bordering on the ocean or on any estuary, creek, river, stream, pond, or lake, or any land under said waters or any land subject to tidal action, coastal storm flowage or flooding." G.L. c. 131 §40. See 310 CMR §10.02(1) (defining areas subject to protection). The terms "bogs," "coastal wetlands," "freshwater wetlands," "swamps," "wet meadows" and marshes are defined in c. 131, §40 primarily in terms of the types of vegetation characterizing such areas.

(the "buffer zone"), which "will alter" a protected wetland area that is "significant" to the wetland interests protected by the Act (e.g., public or private water supply, groundwater supply, or the prevention of pollution). G.L. c. 131, §40; 310 CMR §10.02(2). Activities outside the protected wetland areas or buffer zone are subject to regulation only if and when the activity "actually alters" a protected wetlands area. Id. A project proponent may file a request for a determination of applicability of the Act to particular land or work; such a determination (or a notice of intent) is required for work proposed within the buffer zone. 310 CMR §10.05(3). If the conservation commission determines that the proposed work is not within the Act's jurisdiction (i.e., either that the work is not within a protected area or will not "alter" the wetland in question), the work may proceed unless this negative determination is appealed and overturned. G.L. c. 131, §40; 310 CMR §10.05. Otherwise, the commission must issue an order of conditions regulating the project.

The conservation commission is required to act upon a request for a determination of applicability within 21 days, and must act upon a Notice of Intent within 21 days after the close of a public hearing upon that application by issuing either a negative determination or an order of conditions. G.L. c. 131, §40; 310 CMR §10.05. The commission's determination of applicability or order of conditions may be appealed to the Department of Environmental Quality Engineering ("DEQE") within 10 days of the commission's action. DEQE is required to act upon such appeals within a prescribed time period by issuing, as applicable, either a Superseding Determination of Applicability or a Superseding Order of Conditions. Such superseding orders and determinations by DEQE may be further appealed within the agency by filing a request for an adjudicatory hearing within 10 days of such actions. Id. DEQE's final decision following such an adjudicatory hearing is subject to review in the Superior Court if appealed within 30 days of the agency's decision. G.L. c. 30A, §14.

The Act is implemented by the Massachusetts Wetlands Regulations, 310 CMR §10.00 et seq., which were comprehensively revised by DEQE in

late 1982. The revised regulations became effective April 1, 1983. These regulations define in detail the resource areas and activities subject to regulation under the Act; the procedures to be followed by project proponents, conservation commissions, and DEQE with respect to proposed projects affecting wetlands; and include detailed provisions describing the characteristics, significance, and performance standards for work in particular types of resource areas for both coastal and inland wetlands. Violations of the Act may be enjoined by the Massachusetts courts, and are punishable by criminal penalties. G.L. c. 131, §40.

Inland and coastal wetlands may also be protected from "alter[ation] or pollut[ion]" by inland or coastal wetlands restrictions established by orders of the Department of Environmental Management ("DEM") and recorded in the appropriate registry of deeds. G.L. c. 131, §40A (inland wetlands restrictions); G.L. c. 130. §105 (coastal wetlands restrictions). See 302 CMR §§4.00, 6.00 (DEM wetlands restriction regulations).

2. The Utility Exemption

The Act specifically exempts from regulation activities otherwise subject to the Act's provisions which occur

> in the course of maintaining, repairing or replacing, but not substantially changing or enlarging, an existing and lawfully located structure or facility used in the service of the public and used to provide electric, gas, water, telephone, telegraph and other telecommunications services. . . .

G.L. c. 131, §40 (first paragraph)(emphasis added). Thus, the maintenance of existing electric utility lines is exempt from regulation by conservation commissions and the DEQE under the Act. It appears that this exemption is not applicable to the maintenance of the facilities of railroads which, although regulated as "utilities," are not mentioned in the statute's list of exempted utility structures or facilities.

DEQE also takes the position, based upon the exemption's reference to "existing . . . structure[s] or facilit[ies]," that the exemption is inapplicable to new or proposed power lines, and that it is thus within

the jurisdiction of local conservation commissions and DEQE under the Act to regulate herbicide use on new power lines. See Letter from William J. St. Hilaire, P.E. (DEQE) to Ronald Boches (New England Power Company) accompanying DEQE Superseding Order of Conditions (Amesbury, No. 2-58, September 2, 1982); 310 CMR §10.53(3)(d). Also, DEQE interprets the maintenance exemption as limited to "generally accepted maintenance techniques used by the industry as a whole," which apparently includes the application of generally used herbicides by standard application methods (but not the use of unusually toxic herbicides). See Memorandum from Carl F. Dierker, DEQE Deputy General Counsel, to Robert P. Fagan, regarding herbicide applications by electric utility companies (July 22, 1982).

3. "Alteration"

The critical question in determing the extent of the regulatory authority of DEQE and local conservation commissions over railroad and non-exempt electric utility herbicide applications under the Act is whether the proposed herbicide use will "alter" any of the wetland resource areas protected by the Act. The DEQE regulations broadly define "alter" as "to change the condition of any Area Subject to Protection Under the Act," including for example "the destruction of vegetation" or "the changing of . . . [the] physical, biological or chemical characteristics of the receiving water." 310 CMR §10.04. Thus, any non-exempted application of herbicides in protected wetlands or in the buffer zone which, through drift, runoff, or otherwise, will have any discernible effect upon a protected wetland area is subject to regulation under the Act. Whether alteration will result in a particular case is essentially a scientific question of fact to be resolved in the first instance by the conservation commission and, upon appeal, by DEQE.

For example, in the case of the New England Power Company's proposal to construct new power lines through wetland areas in Amesbury and Groveland, DEQE concluded that "[t]he application of herbicides in this case clearly has potential impact on the protected interests of groundwater quality, protection of public and private water supplies, and prevention of pollution as set forth in the Act," and thus imposed

a number of conditions (substantially incorporating the Pesticide Board's Interim Guidelines) upon the use of herbicides in maintaining the new power line. Letter from William J. St. Hilaire, P.E., to Morris Cherkofsky, accompanying DEQE Superseding Order of Conditions (Groveland, No. 30-22, February 28, 1983), p. 2, Letter from St. Hilaire to Boches, supra, p. 1. In another case, however, DEQE ruled that while a railroad's herbicide application within the buffer zone was subject to the Act, no Notice of Intent was required where DEQE found that spraying would not alter protected wetlands if done subject to specified conditions. Letter from Roland J. Dupuis (DEQE) to Mass. Railroad Association (Palmer, Appeal/Superseding Determination, June 23, 1983). The issue of whether railroad herbicide applications will alter adjacent wetlands is currently before DEQE in adjudicatory appeals involving the towns of Clinton and Leverett.

C. Water Supply and Groundwater Regulation

1. DEQE Water Supply Regulation

In addition to its authority to protect wetlands, DEQE has broad statutory authority to prevent the contamination of public water supplies. DEQE has "general oversight and care of all inland waters and of all streams, ponds, and underground waters used by . . . any person in the commonwealth as sources of ice or water supply and of all springs, streams, and water courses tributary thereto." G.L. c. 111, §159. DEQE has general rulemaking authority to issue regulations and orders "necessary to prevent pollution and to secure the sanitary protection of all such waters used as sources of water supply. . ." G.L. c. 111, §160. DEQE's orders and regulations are judicially enforceable. Id., §164. Chapter 111 also prohibits the discharge "into any stream or pond, or upon their banks . . . or into any feeders of such pond or stream within 20 miles above the point where such supply is taken" of any "polluting matter, of such kind and amount as . . . will corrupt or impair the quality of the water of any pond or stream used as a source of ice or water supply." Id., §167. DEQE is also authorized to make rules and regulations "for the sanitary protection" of waters used by the Metropolitan District Commission ("MDC") for water supply purposes. G.L. c. 92, §17. Chapter 92 declares it unlawful to "corrupt,

render impure, waste or improperly use" any water supply of any town within the Metropolitan District. Id., §18. Violations of this provision are subject to criminal penalties. Id., §22.

Pursuant to these statutory authorities, DEQE has promulgated its Drinking Water Regulations, 310 CMR §22.00, et seq., which set water quality standards and contain numerous prohibitions to protect ground and surface water supplies from pollution. The DEQE regulations prohibit the discharge of any substance which in DEQE's opinion is "poisonous or injurious either to human beings or to animals, . . . directly into or at any place from which such liquid or substance may flow or be washed or carried into said source of water supply or tributary thereto." 310 CMR §22.20(3). This regulation applies to "all land and water courses used as or tributary to a public [surface] water system," with certain limited exceptions. Id., §22.20(1). With respect to groundwater supplies, DEQE may order that the operator of a public water supply acquire at least 250 feet of land (or 400 feet in the case of a gravel-packed well) surrounding a source of groundwater used for drinking water purposes, in order to protect such groundwater supply from contamination, and DEQE "may order greater distances or permit lesser distances . . . if [DEQE] deems such order or permission necessary or sufficient to protect the public health." Id., DEQE apparently believes that it has the ancillary authority to restrict or prohibit activities it deems likely to cause contamination of a groundwater supply within a prescribed "buffer zone" around wells or other groundwater sources. See St. Hillaire letter regarding Amesbury wetlands, supra, p. 2. (citing Drinking Water Regualtions). The DEQE regulations also prohibit the discharge of any "polluting liquid or other substance of a nature poisonous or injurious either to human beings or to animals . . . into any lake, pond, reservoir, stream, ditch, water course, or other open waters, the water of which flows directly or ultimately into any waters" used by MDC for water supply purposes. 310 CMR §24.01. DEQE has taken the position that the above-quoted provisions of its Drinking Water Regulations authorize it to impose conditions upon herbicide use for right-of-way maintenance purposes. See St. Hilaire letter regarding Amesbury wetlands, supra, p. 2.

2. The Massachusetts Clean Waters Act

The Massachusetts Clean Waters Act contains two provisions that may apply to contamination of water resources by herbicides. First, in cases of "discharge of . . . hazardous material into or proximate to any waters of the commonwealth" responsible parties are jointly and severally liable to the commonwealth for investigation and cleanup costs, and/or damages to natural resources. This statute also permits recovery of damages to private property, and imposes criminal penalties for unlawful discharges. G.L. c. 21, §27(14) (repealed on March 24, 1983 by Chapter 7, Acts of 1983, and replaced by G.L. c. 21E, to the same effect).[2] Second, G.L. c. 21, §42 prohibits "the discharge of any pollutant into waters of the commonwealth" without a permit from DEQE, and imposes civil and criminal penalties for violations. However, under recent DEQE regulations effective October 15, 1983, it is unclear whether runoff from herbicide use on rights-of-way requires a discharge permit. Compare 314 CMR §5.05(9) (discharges to groundwater from "right-of-way maintenance activities" exempt from permit requirement), with 314 CMR §3.05 (no such exemption for discharges to surface water).

D. Electric Utility Regulation

The Massachusetts Department of Public Utilities ("DPU") is charged with "the general supervision of all gas and electric companies and shall make all necessary examination and inquiries and keep itself informed as to the condition of the respective properties owned by such corporation and the manner in which they are conducted with reference to the safety and convenience of the public, and as to their compliance with the provisions of law. . . ." G.L. c. 164, §76. The DPU is given broad rulemaking authority to establish regulations that it deems necessary to carry out its statutory duties. Id., § 76C. See Cambridge Electric Light Co. v. Department of Public Utilities, 363 Mass. 474, 494-95 (1973). It has been held that the DPU has "reasonably comprehensive" authority to regulate electric transmission lines,

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2 Chapter 21E, popularly known as the Massachusetts Superfund Act, excludes from its definition of unlawful "releases" of hazardous material the application of pesticides "consistent with their labeling." G.L. c. 21E, §2.

and the safety thereof. Boston Edison Co. v. Sudbury, 356 Mass. 406, 418-20 (1969). No DPU statute was found, however, to specifically authorize the DPU to regulate herbicide use for right-of-way maintenance by electric utilities. Nor does any DPU statute address the subject of vegetation control on utility rights-of-way.

The DPU regulations promulgated pursuant to Chapter 164 include the "Code for the Installation and Maintenance of Electric Transmission Lines," 220 CMR §125. These regulations deal with the design and construction of transmission line structures. No provisions relating to right-of-way maintenance or vegetation control are included among them.

The DPU is required to approve any local ordinances or regulations (pursuant to c. 166, § 25) "affecting the erection, maintenance or operation of a line for the transmission of electricity." G.L. c. 166, §27. However, an opinion of the Attorney General on the subject concluded that local board of health regulations restricting the use of herbicides on utility rights-of-way were not subject to review and approval or disapproval by the DPU pursuant to c. 166, §27. Mass. Atty. Gen. Op. 82/83-12 (May 11, 1983) at 8. (See Part G, infra).

E. Railroad Regulation

As with electric utilities, no government regulation squarely addresses the issue of herbicide use or requires the total eradication of vegetation along railroad rights-of-way. At the federal level, The Federal Railroad Safety Act of 1970, 45 U.S.C. §421, et seq., was enacted "to promote safety in all areas of railroad operations and to reduce railroad related accidents and ... deaths and injuries to persons and to reduce damages to property caused by accidents involving any carrier of hazardous materials." 45 U.S.C. §421. The legislative history of this statute does not indicate that roadbed vegetation was a safety hazard of Congressional concern. Nonetheless, pursuant to this authority, the Federal Railroad Administration has promulgated federal track safety

standards, 49 CFR §213.1, which include the following provision governing vegetation control:

> Vegetation on railroad property which is on or immediately adjacent to roadbed must be controlled so that it does not--(a) become a fire hazard to track-carrying structures; (b) obstruct visibility of railroad signs and signals; (c) interfere with railroad employees performing normal trackside duties; (d) prevent proper functioning of signal and communication lines; or (e) prevent railroad employees from visually inspecting moving equipment from their normal duty stations.

49 CFR §213.37. These regulations, however, do not prescribe that such vegetation be controlled through the use of herbicides or any other particular technique.

The DPU also has broad regulatory authority over railroads, including "general supervision and regulation of, and jurisdiction and control over" railways. G.L. c. 159, §§10, 12; Newton v. Department of Public Utilities, 339 Mass. 535, 541 (1959). Pursuant to this authority, the DPU has promulgated "Railroad Safety Regulations," 220 CMR §150.00, et seq., which include regulations concerning track inspection, track maintenance, and track alterations, but have no provisions concerning vegetation control or right-of-way maintenance. Another statute, G.L. c. 160, §235A, requires all railroads to "keep the full width of all [their] locations, to a point 200 feet distant from the center line on each side thereof, clear of dead leaves, dead grass, dry brush or other inflammable material. . . ." It is questionable whether this statute was intended to require the eradication of live vegetation along railroad rights-of-way, as opposed to the clearing of dead vegetation that has been killed by herbicides or otherwise. No regulations requiring control of vegetation have been promulgated under this statute.

F. State Regulation of New Facilities

1. Energy Facility Siting

Pursuant to the Massachusetts Energy Facility Siting Act, G.L. c. 164, §69G, et seq., electric companies operating in Massachusetts are required to obtain approval from the Energy Facilities Siting Council ("Council") for the construction of major new or expanded transmission facilities (69 kV or more and one mile or more in length). In approving

a proposed facility, the Council must determine that it will "provide a necessary energy supply . . . with a minimum impact on the environment at the lowest possible cost." G.L. c. 164, §69H.

Where a utility is precluded from constructing an approved facility by state or local permit denials or other regulatory obstacles, the Council may upon application issue a "certificate of environmental impact and public need" ("Certificate") with respect to such facility, which in effect constitutes a general composite permit for the proposed facility. G.L. c. 164, §69K. The Council is authorized to prescribe and amend terms and conditions of such a Certificate, including conditions to mitigate or regulate environmental impacts. Id., §69(0). Council Certificates may not override local zoning bylaws in effect before an electric company files for Council approval of a facility. 980 CMR §6.02(2)(f).

The Council's regulations, 980 CMR §1.00, et seq., contain no provisions directly referring to herbicide use. The Council's Administrative Bulletin 78-2, made part of its regulations by action of the Council on December 1, 1978 (EFSC Rule 64.8[3]) requires that a company, before it may construct a new transmission line, describe its planned maintenance practices and provide information concerning surface waters and water courses, aquifers, springs and major wells, wetlands, private on-lot wells, and forest type and vegetation to be cleared. The company must also prepare an environmental assessment of the effects of the proposed transmission line, including a comparison with at least one practical alternative corridor or route. In a 1981 decision, the Council approved, subject to certain conditions, the use of herbicides by a utility on a new transmission line right-of-way in Brewster, Dennis, and Orleans. In re Commonwealth Electric Company, EFSC No. 79-4B (April 3, 1981).

2. MEPA

The Massachusetts Environmental Policy Act, G.L. c. 30, §62, et seq., requires the filing of notices of intent known as Environmental Notification Forms ("ENF") and the preparation and review of Environmental

Impact Reports ("EIR") for projects meeting certain thresholds prescribed in the MEPA regulations, 301 CMR §10.00, et seq. Appendix C to the MEPA regulations, 301 CMR §10.32, lists categorical exclusions and inclusions for determining whether an ENF must be filed and an EIR prepared for a particular project. Generally, these rules require the filing of an ENF, and where indicated the preparation of an EIR, for projects having significant state participation (e.g., through funding or sponsorship), size, and/or environmental impact. There is no general requirement in the MEPA regulations that an ENF or EIR be filed prior to undertaking a particular application of herbicides for railroad or utility right-of-way maintenance. In the case of work undertaken or funded by a state agency (e.g., the Executive Office of Transportation and Construction or the Department of Public Works), "[r]outline maintenance of land, water and vegetation, to insure safety or suitability for the uses to which it is put, . . . and not effecting any substantial change in use" is excluded from the requirement of filing an ENF or EIR, including in particular "[a]pplication of pesticides or herbicides . . . except where a generic environmental impact report is required, has been filed or is in preparation." 301 CMR §10.32(2)(f).

In general, projects undertaken by private parties must file an ENF (and perhaps an EIR, if required by the Executive Office of Environmental Affairs) if they require any state agency permits listed in 301 CMR §10.32(3) and exceed specified size thresholds. It appears that no state permits are per se required for herbicide applications on railroad and utility rights-of-way. However, where DEQE issues a Superseding Order of Conditions for the "alteration" of more than one acre subject to the Wetlands Protection Act or affecting more than 500 feet of "bank" subject to the Act, an ENF is required. 301 CMR §10.32(3)(b)(1). Another wetlands threshold, 301 CMR §10.32(5)(a)(2), requires the preparation of both an ENF and an EIR for "any project requiring alteration of 10 or more acres of land subject to [the Wetlands Protection Act]." This class of categorically included projects would appear to be a subset of those subject to the one acre wetlands threshold, supra, and involves the same type of factual determination as to the

amount of wetlands to be "altered." Note that these MEPA wetlands thresholds do nt include acreage within the buffer zone. 310 CMR §10.07(3). Thus, MEPA may apply to certain herbicide applications that affect wetland resources.

G. Local Regulation and State Preemption

In response to local concerns about potential adverse effects of herbicides on public health and water supplies, a number of cities and towns in Massachusetts have adopted measures that directly or indirectly restrict or prohibit the use of herbicides for right-of-way vegetation control. These local actions have included orders by local conservation commissions pursuant to the Wetlands Protection Act, Board of Health regulations and orders, and various types of zoning or "police power" bylaws, including groundwater protection bylaws, wetlands protection bylaws, and outright herbicide bans or restrictions. The validity of such local enactments, to the extent that they purport to regulate herbicide use, is the subject of considerable controversy. Railroads and electric utilities, among others, have taken the position that such local regulation conflicts with, and is preempted by FIFRA and the MPCA, and that municipalities are precluded from regulating in this area. Litigation involving the validity of local Board of Health regulations restricting or prohibiting herbicide use is currently pending. See Town of Wendell v. Bellotti, C.A. No. 15119 (Franklin Superior Ct.).

1. Specific Statutory Authorities

A number of Massachusetts statutes give cities and towns the power to regulate in various areas that potentially affect herbicide application by electric utilities and railroads. Putting aside the question of preemption, several of these statutory authorities appear broad enough on their face to authorize local regulation affecting herbicide use. These include the following:

First, pursuant to the Wetlands Protection Act, G.L. c. 131, §40, local conservation commissions have the authority to issue orders of conditions which restrict or prohibit activities that will "alter" any

significant wetland resource area. See Part B, supra; Hamilton v. Conservation Commission of Orleans, 1981 Mass. App. Adv. Sh. 1521, 1528.

Second, pursuant to G.L. c. 111, §31, local boards of health have broad powers to adopt "reasonable health regulations," enforced by fines, which regulations must be filed with DEQE. See generally Board of Health of Woburn v. Sousa, 338 Mass. 547, 551-52 (1959).

Third, with respect to electric utilities, G.L. c. 164, § 75 provides that "[t]he aldermen or selectmen may regulate, restrict and control all acts and doings of a[n] [electric company] which may in any manner affect the health, safety, convenience or property of the inhabitants of their towns." However, the significance of this broad, relatively old statute is unclear, since it has been held that more specific, recent utility regulation statutes supersede c. 164, §75 with respect to their subject matter. New England LNG Co. v. Fall River, 368 Mass. 259, 265 (1975).

Fourth, G.L. c. 166, §25 authorizes boards of selectmen to "establish reasonable regulations for the erection and maintenance of all lines . . . for the transmission of electricity" permitted within their towns. G.L. c. 166, §27 (emphasis added). Such regulations may not take effect until approved by the DPU. Id. However, the Attorney General has rendered an opinion that regulations adopted by local boards of health, rather than selectmen, to restrict the use of boards of health, rather than selectmen, to restrict the use of herbicides on utility rights-of-way, are not subject to approval by the DPU pursuant to c. 166, §27, since these regulations were not adopted pursuant to §25. Op. Atty. Gen. 82/83-12 (May 11, 1983).

Fifth, pursuant to G.L. c. 40, §§39A-E, towns may acquire (subject to DEQE approval) lands and waters for water supply purposes, and anyone who "willfully or wantonly corrupts, pollutes or diverts" any such waters is subject to suit by the town for treble damages, as well as criminal penalties. Id. §39G.

Sixth, under the Zoning Act, G.L. c. 40A, §5, cities and towns are authorized to adopt zoning ordinances, which may "regulate the use of land . . . to the full extent of the independent constitutional powers of cities and towns to protect the health, safety and general welfare of their present and future inhabitants." Id., §1A.

Finally, cities and towns are authorized to adopt general "police power" bylaws (i.e., to protect the public health, safety, welfare, and convenience) for any purpose not inconsistent with the state law. G.L. c. 40, §21; c. 43B, §13. (See discussion of Home Rule, infra). Both general and zoning bylaws are subject to approval by the Attorney General before they may take effect. G.L. c. 40, §32; see c. 40A, §5.

2. Home Rule Powers

The powers of Massachusetts cities and towns to enact local ordinances and bylaws were fundamentally broadened in 1966 by the adoption of the Home Rule Amendment, Mass. Const., Art. 89, §6, which allows municipalities to adopt any local ordinance or bylaw which is "not inconsistent with" the Massachusetts Constitution or statutes. See also the Home Rule Procedures Act, G.L. c. 43B, §13. By virtue of the Home Rule Amendment, cities and towns are no longer limited, as they formerly were, to adopting local legislation only on subjects specifically authorized by the legislature, pursuant to G.L. c. 40, §21. Thus, the fundamental inquiry in determining the validity of a local regulation is not whether it is "inconsistent with" state (or federal) law and thus preempted. See generally Jerison, Home Rule in Massachusetts, 67 Mass. L. Rev. 51 (1982).

3. The Preemption Doctrine

As just noted, the Home Rule Amendment permits municipalities to regulate only in ways "not inconsistent with" state law. In areas in which there is state legislation, local bylaws or ordinances "will be deemed void if they are inconsistent with any portion of the General Laws." Beard v. Salisbury, 378 Mass. 435, 440 (1979). The same is true in the case of any conflict between state or local enactments and federal law. See e.g., Florida Lime and Avocado Growers v. Paul, 373

U.S. 132, 146-47 (1963). Thus, state and federal legislation preempts local legislation in any area in which the two conflict, and the legislature clearly has the power to "restrict local legislative action or denying municipalities power to act at all." Arlington v. Board of Conciliation and Arbitration, 370 Mass. 769, 773 (1976).

The leading case on the subject of state preemption is Bloom v. Worcester, 363 Mass. 136 (1973). In explaining the standards for determining whether a town bylaw is "inconsistent with" state law, the Supreme Judicial Court in Bloom stated that the test is whether the Legislature intended to preempt local action on a particular subject, and that "[t]he legislative intent to preclude local action must be clear." 363 Mass. at 155. Where there is no explicit indication of the legislature's intention in this respect, however, an intention to preempt local action may be inferred from factors including the existence of comprehensive legislation on a subject which effectively occupies the field, and specific statutory provisions describing what municipalities can and cannot do, or limiting the manner in which cities and towns may act on the subject. Id. at 155-56. Where the operation of a local ordinance "will in any way frustrate the achievement of any statutory purpose," the ordinance is invalid. Id. at 155, 158. Conversely, "[i]f the State legislative purpose can be achieved in the face of a local ordinance or by-law on the same subject, the local ordinance or by-law is not inconsistent with the State legislation, unless the Legislature has expressly forbidden the adoption of local ordinances and by-laws on the subject." Id. at 156.

Thus, where an ordinance or bylaw (1) directly conflicts with the express provisions of a statute, (2) frustrates the purpose of a statute, or (3) attempts to regulate in an area fully and comprehensively regulated by state law, it will be held invalid. See e.g., New England LNG Co. v. Fall River, 368 Mass. 259, 265-67 (1975) (comprehensive state regulation of gas companies preempted local ordinance on subject); Del Duca v. Town Administrator of Methuen, 368 Mass. 1, 10-12 (1975) (comprehensive state legislation, "mandatory in its terms," "describing in detail what municipalities can and cannot do," on subject of planning

boards preempts town bylaw regulating terms of office and powers of broad); Beard v. Salisbury, 378 Mass. 435, 440-42 (1979) (state earth removal statute preempts town bylaw prohibiting exportation of sand and gravel excavated within the town); Rogers v. Provincetown, 1981 Mass. Adv. Sh. 1728 (state statute authorizing operation of mopeds on any public way preempts town bylaw prohibiting rental of mopeds). On the other hand, where the local ordinance is wholly consistent with the purpose of state legislation on the subject and furthers its purpose, the local regulation will be upheld even it is more stringent or broad than the state statute on that subject. See e.g., Bloom v. Worcester, supra, 363 Mass. at 159-60, 163 (local human rights commission established by city ordinance consistent with purpose of state anti-discrimination legislation); Lovequist v. Conservation Commission of Dennis, 379 Mass. 7 (1979) (local wetlands protection bylaw not inconsistent with Wetlands Protection Act).

4. Preemption of Local Herbicide Regulation

There is limited Massachusetts judicial precedent on the issue of whether local ordinances or bylaws which purport to restrict in various ways the application of herbicides on railroad and utility rights-of-way are preempted by FIFRA, the Massachusetts Pesticide Control Act, or any of the other state statutes discussed above. The Attorney General has disapproved bylaws adopted by the Town of Wendell which restricted and imposed conditions upon the use of herbicides in various ways, on the ground that such local regulations of pesticides were inconsistent with the MPCA, FIFRA, and the state and federal regulations promulgated pursuant thereto. Letter from Henry F. O'Connell, Assistant Attorney General, to Town Clerk of Wendell (November 19, 1980). The validity of this disapproval is now being litigated. Wendell v. Bellotti, C.A. No. 15119 (Franklin Superior Ct.).

The Attorney General has likewise disapproved similar bylaws variously regulating the use of herbicides and other pesticides adopted by the Towns of Ashburnham (1979), Bellingham (1980) and Orleans, Leyden, Leverett, and Wendell (1981). However, in 1983, the Attorney General approved a bylaw adopted by the Town of Wayland which prohibits

applications of pesticides (including herbicides) by private parties which come into contact with the persons or property of others, unless advance written permission has been obtained. See Letter from Henry F. O'Connell to Town Clerk of Wayland (February 11, 1983). In the 1983 advisory opinion cited supra, the Attorney General noted that while local board of health regulations restricting herbicide application were not subject to DPU approval, such regulations "might be unenforceable on other grounds which are beyond the scope of this opinion," citing analogous cases in which local regulations of utility activities were held invalid. Op. Atty. Gen. 82/83-12, at 9, fn. 4 (May 11, 1983). The latter issue was not decided, however.

Decisions in other jurisdictions have held that local regulation of herbicides is preempted under either FIFRA or state pesticide statutes. See e.g., Town of Salisbury v. New England Power Co., 437 A.2d 281 (N.H. 1981); Long Island Pest Control Association v. Town of Huntington, 341 N.Y.S.2d 93 (N.Y. Sup. Ct. 1973), aff'd, 351 N.Y.S.2d. 945 (1973). Likewise, the legislative history of amendments to FIFRA and the regulations pursuant thereto suggest that Congress did not intend to permit local regulation of pesticides. See, e.g., S. Rep. No. 838, 92nd Cong., 2d. Sess., reprinted in 1972 U.S. Code Cong. & Ad. News 3993, 4066; 40 F.R. 11700 (March 12, 1975).

Nevertheless, DEQE appears to have taken the position that local conservation commissions can restrict utility and railroad herbicide use pursuant to their powers under the Wetlands Protection Act where such herbicide use is likely to "alter" protected wetland resource areas. See Amesbury and Groveland Superseding Orders of Conditions and accompanying DEQE letters, supra (regarding proposed power lines). DEQE did not, however, consider the preemption issue in these cases. Moreover, the Lovequist case, supra, suggests that municipalities could restrict herbicide use as part of a comprehensive wetlands or aquifer protection general or zoning bylaw adopted independently of the Wetlands Protection Act. However, the above-cited precedent suggests that at least certain types of local regulation purporting to restrict or prohibit the use of herbicides may be preempted by FIFRA and the

MPCA, even if such bylaws are consistent with the Wetlands Act. In sum, it is fair to say that the extent and manner in which cities and towns can regulate herbicide use by railroads and utilities for right-of-way maintenance is an unresolved issue.

Unresolved Legal Issues

A. Scope, Interface, and Possible Conflicts Among State Agency Jurisdiction

In addition to the U.S. EPA's jurisdiction to regulate pesticides under FIFRA, state agencies including DEQE (under the Wetlands Protection Act, water supply statutes, and drinking water regulations); the Pesticide Board (under FIFRA, the MPCA, state pesticide regulations and interim guidelines); the DPU (under several railroad and electric utility regulation statutes and regulations); and the Siting Council (under the Siting Act and regulations) all have varying degrees of potentially overlapping regulatory jurisdiction over utility and railroad herbicide application. What potential conflicts or gaps (e.g., concerning groundwater protection) are inherent in the current statutory scheme? Which authority prevails in the event of a conflict?

B. Authority of Cities and Towns to Regulate

Cities and towns, through their conservation commissions, boards of health, and by the adoption of general zoning bylaws, have adopted or have the potential to adopt ordinances, bylaws, regulations, and orders which purport to restrict or prohibit herbicide applications by railroads and utilities in various ways. These local actions may take the form of general prohibitions on herbicide use, facially neutral regulations which may have the effect of restricting or prohibiting herbicide use (e.g., wetland or aquifer protection bylaws), or site-specific restrictions on herbicide application (e.g., wetlands orders of conditions). To what extent are such local regulations preempted by state or federal law? Does the form of the local regulation or bylaw matter?

C. Possible Federal Preemption of State Herbicide Regulation

To what extent is Massachusetts constrained by FIFRA or other federal law in adopting legislation or regulations concerning herbicide use for

right-of-way maintenance? What types of state regulation, if any, would be preempted by FIFRA?

CHAPTER 2. LOCATION OF PUBLIC AND PRIVATE WELLS

Applicators need to know the location of private and public wells that are adjacent to or within rights-of-way. This section discusses possible sources of information on the location of these wells.

Private Wells

The majority of private wells have not been mapped by any state or local agency. . A Massachusetts law, St. 1962 c. 513, requires well drillers to report the location of drilled wells to the Water Resources Commission. This law has been in effect for over 20 years but has not been enforced. Although town-by-town files are currently maintained in the Department of Environmental Management, very few well drillers (considerably under 25%) have complied with the law. If it had been enforced, the compiled data base would have consisted of

> . . . the name of the owner of the well, the geographic location of the well (this shall be given accurately to enable easy plotting on a U.S. Geological Survey Topographic (1:25,000 scale) Map), well depth, depth to bedrock or refusal, casing type, casing size and casing length, well screen type, well screen length, and well screen depth set, static water level, method used to test well yield, length of time (in hours) well pumped, drawdown, well yield, and drilling logs describing the material penetrated.

Future environmental impact assessments would benefit greatly from renewed efforts to enforce this law.

Individual towns have historically required information on the location of wells in the construction of new homes to ensure sufficient distance between wells and septic systems. Some towns have kept records of the locations of the wells; others have merely reviewed the site plans and then filed the permits. Poor record-keeping has been the rule rather than the exception, however; most of the towns contacted in this study say that the locations of wells of new homes have been adequately recorded only in the last two to five years. Information on the wells of homes built prior to that time is essentially non-existent.

Individual homeowners must therefore provide the information on private wells. It has been suggested that applicators conduct a survey of these homeowners in order to map the wells. This, however, would involve considerable time and effort on the part of the applicators, who often do not have the necessary resources. Utility and railroad companies may be able to conduct such a survey if the owners of abutting parcels can be identified. These property owners can be asked to mark the location of their wells on hand-drawn maps or on maps provided by the uitility or railroad companies.

An alternative way to gather this information would be for the towns to send requests for the information to individual homeowners. These mailings would request abutters of rights-of-way to identify themselves and to mark the location of these wells on maps provided by the town, or on hand-drawn maps of the homeowners' properties. This information could then be compiled by the town and forwarded to the applicator, and/or utility or railroad.

This process would take a considerable time to complete. In the meantime, it should be supplemented by an approach used in Vermont. In that state, as well as our own, the utilities and railroads are required to notify the public of impending herbicide applications. In Vermont, however, the notification must also contain a request for landowners to supply information to the utility or railroad on the location of any private well within 100 feet of the right-of-way. The results have not been encouraging, as suggested by the experience of the state highway department when notifying the public of herbicide spraying on highway rights-of-way: only 12 phone calls have been received in 3 years. This approach should therefore be used only as a short term supplement to a more thorough systematic mapping by towns.

Public Wells

Information on the location of public wells is relatively easy to obtain. In Massachusetts, public wells have been mapped by the Department of Environmental Quality Engineering. These maps can be obtained by calling the district offices: Central (617-727-0886), Northeast

(617-935-2160), Southeast (617-727-1440), and Western (413-549-6442). This information can then be transferred to the maps used by the applicators.

It is recommended therefore that:

1. Information on public wells in the Commonwealth of Massachusetts should be obtained from the Department of Environmental Quality Engineering.
2. Information on private wells should be provided by property owners and assembled by towns or by utility or railroad companies.
3. Notifications of impending applications should request information from landowners on the locations of their wells.

CHAPTER 3. BIOLOGICAL CONTROL FOR RIGHTS-OF-WAY: ADDITIONAL DISCUSSION*

The first step to be taken in exploring biological control in any ecosystem should be to study the ecology of the community. This is true for two reasons: (1) Biological control strategies are not necessarily as obvious as standard ones, and must sometimes be developed from a knowledge of the basic biology of the system; and (2) the ramifications of control strategies must be worked out as completely as possible before trying them out. There are many cases in the literature of control attempts that backfired because the behavior of a given species was ignored or because ecological principles were not understood (DeBach, 1974).

The most important ecological principle to be considered here is succession, that refers to the changes over time in species composition of a community, usually in a somewhat predictable order. In northeastern United States forests, this usually refers to sequences of species which colonize open habitats following disturbances, for example agricultural fields and pastures left unmanaged, tree blowdowns, and fires. Roughly, the sequence starts with annuals and grasses, progresses through perennial herbs to bushes to early successional trees, leading finally to the climax vegetation, which in must of Massachusetts is probably oak, white pine, and hemlock (Bromley, 1935).

The rights-of-way problem is to keep forest succession from progressing. Strategies for achieving this end must be based on an understanding of the forces driving succession. Current theories of succession can be divided into two groups. In one group succession is reasonably directional and, therefore, predictable . . . It results from modification of the physical environment by the community; that is, succession is community-controlled even though the physical environment determines the pattern, the rate of change, and often sets limits

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as to how far development can go . . . It culminates in a stabilized ecosystem . . ." (Odum 1969)

In the other group of theories, it is thought that succession does not always go in one direction and can be slowed down or stopped along a successional sequence. According to an alternate explanation of succession put forth by Drury and Nisbet (1973), "most of the phenomena of succession can be understood as consequences of differential growth, differential survival (and perhaps also differential colonizing ability) of species adapted to growth at different points on environmental gradients. The appearance of successive replacement of one "community" or "association" by another results in part from interspecific competition which permits one group of plants temporarily to suppress more slowly growing successors . . . A comprehensive theory of succession should be sought at the organismic or cellular level, and not in emergent properties of communities."

Frank Egler (1954a), who has done a large amount of work on rights-of-way management, termed the first group of theories "relay floristics," in which each successional stage prepares the way for the next. He suggested that operating concomitantly with this is a second factor, the "initial floristic composition." According to this hypothesis, all or almost all of the species are present when succession starts or invade very early on, and "development unfolds from this initial flora, without additional increments by further invasion" (Egler, 1954a)

The three views of succession described above lead to three ideas on control. By the first view, any attempts at keeping trees out of a naturally forested ecosystem must always fight an inexorable process. Trees will always attempt to invade grasses, herbs, or bushes, and so constant surveillance and periodic treatments to kill tree seedlings are necessary. The second view, that of Drury and Nisbet (1973), suggests the possibility that, with a knowledge of the biology of individual species, it may be possible to manipulate natural ecosystems "off the main track" of succession. Third, a restatement of the theory of initial floristic composition is that one important factor in succession is

"getting there first." Plants have a higher chance of succeeding if they have advance reproduction. For example, tree species with seedlings which can remain in the understory of a forest will have a higher chance of moving into the canopy when a large tree falls than tree species which cannot tolerate shade as seedlings. By this view, selectively destroying the appropriate tree species early in succession may lead to a relatively stable community which is resistant to tree invasion.

Thus, based on an understanding of the ecology of forest succession, biological control for rights-of-way is different from other biological control situations. The goal in most cases is to limit or eradicate a given species from a given type of environment; for example, to reduce the populations of gypsy moth in forests of the northeastern United States. This is usually accomplished by adding to the ecosystem an antagonistic species; for example, the nucleopolyhedrosis virus for control of gypsy moth. However, adding a non-native species to a right-of-way is extremely dangerous, because of the possibility that the controlling agent will affect the forest outside the right-of-way. Here, the problem is to create a specific pattern of distribution among existing species, and in most cases this means keeping a thin strip of land through forest (or what would become forest if left alone) in an early successional state. Instead of trying simply to reduce the density of target species, it is more useful to approach the problem as one of establishing a community of competitors that can keep trees from invading.

Mechanism of tree inhibition by shrub and herbaceous communities

The next step is to explore the possible mechanisms by which treeless plant communities could exclude trees. In this section, proposed mechanisms are reviewed. In the next, examples of communities stable to tree invasion are given.

Several mechanisms have been proposed to explain how dense shrub, grass, forb, or fern communities can prevent or reduce tree seedling growth. No case is fully understood. Competition for light, nutrients,

and moisture is probably important to some degree in all or most cases (Niering and Goodwin, 1974). At least in some cases, the desired species are able to outcompete tree seedlings if they are given a head start. There are three ways in which plants can invade open areas quickly. Plants such as ferns and some weeds have seeds that are numerous, tiny, and wind-dispersed. Another mechanism is vegetative spreading, that is, without seeds. Bramble and Byrnes (1982) found that most of the species in their stable communities spread by sending up shoots from rhizomes, underground stems that run horizontally. Once a single plant is established from seed it can spread to cover an area with dense growth which may outcompete any other seedlings within its borders.

The third way to get a head start is to have shade-tolerant seedlings in the forest understory, which can enter the canopy as soon as a space is made available. Horsely and Marquis (1983) described studies of Grisez and Peace (1973), in which the presence of advance reproduction was found to be the most important factor determining whether forest regeneration occurred on clear-cut Pennsylvania lands.

Soil conditions are extremely important determinants of the success of plant species. Horsley (1977a) describes clear-cut areas in Pennsylvania that failed to regenerate, due at least in large part to fires which destroyed much of the organic matter of the soil and to poor drainage along stream bottoms or in high flats underlain by fragipans, (hard, impermeable underground layers). Niering and Goodwin (1974) suggested that heaths, (communities of ericaceous shrubs such as blueberry and huckleberry) may inhibit tree establishment by buildup of acid duff. Also, preliminary data of Warren and Niering (1973), as reported by Niering and Goodwin (1974), suggest that moisture stress under huckleberry clones may be important. Bramble and Byrnes (1982) in their study of a Pennsylvania right-of-way, concluded that "while it appears that there are cases where certain shrub communities are relatively permanent and highly resistant to tree invasion, these usually occur under special conditions of habitat

such as sites highly unfavorable for trees, or where human and animal disturbance and fire are continuous."

Grazing can affect species composition of plant communities. Horsley and Marquis (1983) concluded that deer browsing affected growth of certain species of trees as well as blackberry and raspberry in clear-cut areas of central Pennsylvania. Little bluestem grass (Andropogon scoparius), which may be stable to tree invasion (Niering and Goodwin, 1974), is destroyed by grazing (Bromley, 1935).

Fire is also an important determinant of species composition. Many species that are desirable for right-of-ways, such a little bluestem, are common invaders of burned-over areas (Swan, 1970; see also chart below).

Finally, allelopathy has been implicated in resistance to tree establishment (Horsley, 1977a and b). Allelopathy is the production by a plant of chemicals inhibitory to another. Horsley studied the failure of certain areas in Pennsylvania to reforest following clear-cutting. He found that washings from goldenrods, asters, and ferns inhibited seed germination and seedling growth of black cherry.

In summary, many mechanisms have been considered to account for the inhibition of tree establishment by certain plant communities. Some factors, such as soil type, are likely to be of some importance in all cases. Other factors, such as allelopathy, have been implicated by experimental evidence in specific cases. At this point, it is impossible to describe definitively and completely the mechanisms of inhibition in any given case.

Examples of Plant Communities Stable to Tree Invasion

The next question to be asked is: Are there plant communities where competitors that resist tree invasion have been established, either purposefully or fortuitously? The answer for the northeastern United States is yes, both purposefully and fortuitously.

There are several examples of plant communities established by various types of environmental perturbations, that simply are not invaded by trees. Niering and Egler (1955) reported a stand of Viburnum lentago in southwestern Connecticut, considered to have arisen fortuitously on an old pasture, which had no tree invasion for at least 25 years. Niering and Goodwin (1974) mentioned communities of witch hazel, speckled alder, sheep laurel and other species in various parts of Connecticut which appeared stable to tree invasion for up to several decades.

Foresters have long noted that certain shrubs, herbs and grasses can inhibit forest regeneration. Their problem is a potential boon for rights-of-way management. For example, Horsley (1977a and b) described areas in northwestern Pennsylvania, clear-cut and burned fifty years previously, which still had no or little tree regeneration. These areas had dense ground cover dominated by grasses, goldenrod, aster, and ferns. In the southeastern United States, forest regeneration on some three million acres of land has been prevented by thickets of rhododendron and mountain laurel (Wahlenberg and Doolittle, 1950, McGee and Smith, 1967).

There are also examples of rights-of-way, under various management schemes, on which communities stable to tree invasion for up to several decades have been established. Niering and Goodwin (1974) selectively sprayed a power line right-of-way in Connecticut in 1953; periodically they re-treated the area to root-kill small amounts of new reproduction and trees initially missed. In 1970, the line was reconstructed and trees within the right-of-way were given a basal treatment. These treatments resulted in a mosaic of relatively stable shrub communities and less stable herblands. Areas of continuous dense shrub cover resisted tree establishment for at least 15 years. The major shrub species involved were smooth alder, coast pepperbush, winged sumac, northern arrowwood, blackberry, greenbrier, and hayscented fern. Very little invasion occurred in clones of blueberry (Vaccinium vacillans), huckleberry, and greenbrier. In addition, pure stands of little bluestem grass showed remarkable stability. The authors

recommended that "in view of the stability of shrub communities and of the possibility of encouraging them through the selective removal of tree growth, the potential for creating shrub cover in vegetation management is great."

Bramble and Byrnes tested various tree-removal methods on a power line right-of-way in Pennsylvania. The line had been cleared in 1951-52 and was sprayed in 1953. Follow-up basal treatments were given in 1954 and 1966. Spot cutting and stump spraying were carried out in 1978-79 to control the tallest trees. In 1980 and 1982 selective basal spray was applied. They found that a dominant shrub cover interspersed with herbaceous openings developed on all treatment areas over 30 years. Some of the major species were the same as those found by Niering and Goodwin (1974).

However, in contrast to Niering and Goodwin (1974), Bramble and Byrnes (1976) stressed that some shrub species were resistant to tree invasion and others were not; that is, the life form (for example, shrub vs. herb vs. grass) was not as important as the particular species involved. They found that the patches with fewest invading trees in 1976 had low early blueberry, bear oak, meadow fescue, and mixtures dominated by fescue and narrow leaved goldenrod. The patches with the heaviest tree seedling densities were huckleberry, rough goldenrod and blackberry, as well as mixtures dominated by rough goldenrod, hayscented fern, sweet fern, and blackberry. The dominant community type throughout the thirty years was composed primarily of bracken fern, sedge, loosestrife, and blueberry; in 1976 this community covered 27% of the right-of-way and had the second highest density of trees emerging above the group cover.

Bramble and Byrnes (1982) concluded that "areas that remained absolutely stable on the right-of-way over the thirty years were rare indeed". It appeared that what seemed to be stable communities were in fact mosaics of cyclic changes operating at the local level. There was a constant trend of the fern, grass, herb, and shrub vegetation towards development of a dominant shrub cover.

In 1934-36 a fire line was cleared and harrowed through a southeastern New York forest. Most of the line developed a brush community, including trees. One part produced a complex of stable treeless communities which until at least 1953 resisted tree invasion (Pound and Egler, 1953). The communities included ferns, sedge, and bushes.

Horsley (personal communication; September 8, 1983) described a power line right-of-way in Pennsylvania that had been sprayed once 40 years ago and on which a community of ferns developed. Tree seedlings did germinate in the community, but the seedlings did not emerge above the fern cover.

One must be extremely careful when comparing plant community dynamics of different regions, because of differences in temperature, rainfall, soil types, and species composition. None of the above cases is in Massachusetts. However, most of the important species mentioned in the studies do occur in Massachusetts. Species that are problematic in other regions may not be so in Massachusetts. Also, the resistances of plant species to tree invasion may change at different latitudes.

The set of forces driving community dynamics are likely to be the same throughout the northeastern United States, although the relative importances of them will vary region to region. By studying cases in different regions, one can learn under what conditions what forces are important and use this information to extrapolate to the region of interest.

In summary, several examples of arrested or retarded succession are known in the northeastern United States. The communities arise in these areas either fortuitously, through selective herbicide use and mechanical control, or following logging and burning. Several species are found in more than one community. There are no absolutely stable communities, only some communities with relatively greater stability than others. Although Niering and Goodwin stress the stability of shrub clones, they, along with Bramble and Byrnes, also stress the concept of relative stability, as opposed to the idea of climax in classical

succession theory. In addition, life form is not a sufficient indication of resistance to tree invasion. The particular species involved must be considered.

Species associated with the inhibition of tree invasion The following provides a brief description of a few of the species that were found to be components of stable communities that resisted tree invasion over time.

Andropogan scoparius (little bluestem) is a grass that can tolerate a variety of conditions, including open woods, pinelands, dry clearings, prairies, and open rocky areas on hilltops. It forms a deep root system that can limit the invasion of all trees except those that have a deep initial taproot. The dense, fibrous root system successfully competes with trees for moisture, particularly at lower soil depths. Andropogan often dominates old fields and can form a thick cover, particularly if burned (Bromley, 1935; Jorgensen, 1978; Richards, 1973).

Dennstaedtia punctilobula (hay-scented fern) is a fern that will grow in a rocky or low-nutrient soils in wet or dry conditions. It can form dense colonies in pastures and roadsides. It was found to inhibit tree invasion in an Allegheny Plateau forest in Pennsylvania after trees were cut (Cody et al., 1977; Horsley, 1977b).

Gaylussacia baccata (huckleberry) is a shrub that grows in a variety of conditions such as dry or moist woods, thickets, clearings, and swamps. It is favored by fire and can be cultivated by seed. On a right-of-way in Connecticut, it was found to be a part of a stable shrub community, forming dense clones. It was also part of a right-of-way community where it was found to be one of the species most resistant to tree invasion (Fernald, 1950; Petrides, 1972; Niering and Goodwin, 1974; Egler, 1954b).

Hamamelis virginiana (witch hazel) is a shrub found in dry or moist woods. It can persist for many years by means of basal suckers,

but expansion requires reseeding. In East Haddam, Connecticut, it was present as thickets in abandoned pastures which were found to be stable for 40 years. It has also been found in stable shrub communities in southeastern New York and on a right-of-way in Connecticut. On a central Pennsylvania right-of-way, it was found to be sparse but consistently present, forming a shrub border at the forest edge (Niering and Goodwin, 1974; Bramble and Byrnes, 1972; Pound and Egler, 1953; DeSteven, 1982).

Kalmia angustifolia (sheep laurel) is a shrub found in old pastures, rocky hilltops, barrens, bogs, open woods and in wet or dry soils. It is often found in areas with a history of forest fire. In spruce-fir forests of Newfoundland, it has been found to form a dominant cover after fires, resisting tree invasion almost indefinitely. It may also be allelopathic, since a water-soluble extract has been found to inhibit root growth of black spruce. In Marlborough, Connecticut, pure clones have been found to be stable to tree invasion for several decades (Jorgensen, 1978; Niering and Goodwin, 1974; Fisher, 1977).

Pteridium aquilinum (bracken fern) is tolerant of both shade and full sun, and can grow in infertile, sandy, and acidic soils, as well as in woods, old pastures, and burned-over areas. Among foresters, it is considered to have the ability to inhibit reforestation, and has been found on several rights-of-way that are stable to tree invasion (in Connecticut, New York, and Pennsylvania). It is considered allelopathic to black cherry and a number of other trees. (Horsley, 1977a; Richards, 1973; Pound and Egler, 1953; Neiring and Goodwin, 1974; Bramble and Byrnes, 1972).

Solidago spp. (goldenrods) are herbaceous plants that are commonly found throughout much of Massachusetts in clearings, along roadsides, and on the borders of woods and streams. Several studies have indicated that it may be allelopathic to trees, including black cherry, yellow poplar, and sugar maple. It responds well to cultivation and can be encouraged by the addition of nutrients (Horsley, 1977a; Richards, 1973; Fisher et al., 1978; Goode, 1980).

Vaccinium spp. (blueberry). In Massachusetts, blueberry grows in poor acid soils of pastures, in rocky areas, swamps, under oak canopies, and other habitats. Low blueberry has been found in stable shrub communities in Connecticut and Pennsylvania. In Pachaug State Forest in Connecticut, attempts to reforest large tracts of land have been thwarted by high densities of blueberry. The only regular care given most blueberry fields is burning every 2 or 3 years, although fertilization promotes their growth (Thomson, 1977; Niering and Goodwin, 1974; Bramble and Byrnes, 1976).

Rubus allegheniensis (blackberry) is a shrub found in dry clearings and thickets, although it grows best in moist, rich soil in open woodlands, and along fences and roadsides. It is a component of stable vegetative communities in Connecticut, southeastern New York, and central Pennsylvania. It has been recommended as an appropriate crop for rights-of-way (Niering and Goodwin, 1974; Bramble and Byrnes, 1976; Duncan, 1935; Goodland, 1973; Pound and Egler, 1953).

TABLE I-1

SPECIES IMPLICATED IN INHIBITION OF TREE ESTABLISHMENT

Common name	Scientific name
Bushes	
Alder, smooth	Alnus serrulata
Alder, speckled	Alnus rugosa
Arrowwood	Viburnum recognitum
Blackberry	Rubus allegheniensis
Blueberry, late low	Vaccinium angustifolium
Blueberry, early low	Vaccinium vacillans
Blueberry, highbush	Vaccinium corymbosum
Dogwood, gray	Cornus racemosa
Greenbrier	Smilax rotundiflora
Hazelnut	Corylus spp.
Honeysuckle, Japanese	Lonicera japonica
Huckleberry	Gaylussacia baccata
Juniper, common	Juniperus communis
Loosestrife	Lysimachia quadrifolia
Meadowsweet	Spiraea latifloia
Mountain laurel	Kalmia latifolia
Nannyberry	Viburnum lentago
Pepperbush, coast	Clethra alnifolia
Sarsaparilla, wild	Aralia nudicaulis
Scrub oak	Quercus ilicifolia
Sheep laurel	Kalmia angustifolia
Sumac, winged	Rhus copallina
Sweet fern	Comptonia peregrina
Teaberry	Gaultheria procumbens
Witch Hazel	Hamamelis virginiana
Ferns	
Bracken fern	Pteridium aquilinum
Hay-scented fern	Dennstaedtia punctilobula
New York fern	Thelypteris (Dryopteris) noveboracensis
Grasses	
Little bluestem grass	Andropogon scoparius
Fescue, red	Festuca rubra
Grass	Calamagrostis cinnoides
Grass, short husk	Brachyelytrum erectum
Panic grasses	Panicum spp.
Sedge, swamp	Scirpus cyperinus

Sedge	Carex crinita
Sedge, vernal	Carex pensylvanica
Upland rice grass	Oryzopsis asperifolia

Forbs

Aster	Aster spp.
Fireweed	Erechtites hieracifolia
Flat pea	Lathyrus sylvestris
Goldenrod, Canadian	Solidago canadensis
Goldenrod, grass-leaved	Solidago graminifolia
Goldenrod, wrinkled	Solidago rugosa

CHAPTER 4. A METHOD FOR MARKING RIGHTS-OF-WAY

A method is needed for marking areas that should not be sprayed on rights-of-way. Zones around wells or wetlands, for example, need to be identifiable to an applicator. The most reliable way to mark areas not to be sprayed is by identifying them on maps carried by the applicator as he moves along the right-of-way. Maps are more reliable than physical markers, which can deteriorate or be vandalized or obscured by vegetation. Physical markers on the rights-of-way should supplement the use of a map. Particularly sensitive points such as wells located within rights-of-way should be marked with a painted metal stake or a wooden stake treated to retard degradation. Additionally, "Warning" markers should be placed on the rights-of-way to give advance notice to an applicator that the segment he is approaching contains an area that should not be sprayed. Advance warning is particularly important on railroad rights-of-way, where the vehicle applying the herbicide may approach a sensitive area suddenly.

A simple system to provide this advance warning would be to use colored signs that would tell the applicator when he was approaching or leaving a sensitive segment. On a utility rights-of-way, these signs (possibly colored metal plates) could be placed on transmission line structures. Different colors would indicate "approaching" or "leaving" a sensitive segment. Each structure that bordered a sensitive area would have two signs, one of each color on each side, so that the segment could be approached in either direction.

On railroad rights-of-way, the same system would be used with the signs attached to the poles running along side the tracks which carry the communication lines. The signs would be placed a short distance away on either side of an area not to be sprayed (e.g. 100 feet). Again there would be a rule for colors indicationg entering and leaving the segment, and again two signs of different colors would be on each pole marking the edge of the segment.

Further consideration of these problems are needed by structural engineers and others able to design a system that is easily maintained over long periods and that does not interfere with railroad or utility right-of-way function or safety.

CHAPTER 5. SPILL CLEANUP

The following information is intended to provide examples of ways to minimize adverse effects of pesticide spills and the disposal of unused material and pesticide containers. This discussion is not meant to provide adequate information to those responsible for reacting to a spill of pesticide material. Additional information can be obtained from the Pesticide Board of the Massachusetts Department of Food and Agriculture.

Spill Cleanup

Before a spill occurs, a contingency plan should be prepared and a clean-up kit should be assembled. Applicators should be familiar with the contingency plan, which should include specific procedures to be followed for liquid and dry herbicides, phone numbers for emergency services, and names of persons to be notified. Spill kits should include copies of the contingency plan, along with such items as a 5-gallon drum, protective clothing, a large quantity of absorbant material (such as sawdust, a floor-sweeping compound or vermiculite), a tarp for covering the spill, first aid material, a shovel, and plastic bags.

When a spill occurs, after any injuries have been attended to, the following steps should be taken:

1. Liquids should be prevented from spreading by trenching or diking the area with absorbant material or inert materials such as sand.

2. The spill should be covered. If it is dry, a tarp can be placed over it or a light sprinkling of water can be applied. Liquids should be covered with absorbant material.

3. Dry spills should be removed by sweeping up small areas at a time, gradually unrolling the tarp to uncover areas to be swept. For liquid spills, the absorbant should be worked into the spill to assure that it thoroughly soaks up the liquid.

4. Dry material or the absorbed liquid should be placed in heavy duty plastic bags or other leakproof containers and labeled.

5. The soil under the spill should be dug to a depth of at least three inches below the depth to which the liquid penetrated. Contaminated soil should be placed in leakproof drums and labeled.

6. All material should be disposed of in an approved hazardous waste disposal facility.

Specific information on spill cleanup for individual herbicides can often be obtained from the material safety data sheet provided by the manufacturer. For instance, the recommendations for spill clean up provided by Dow Chemical U.S.A. for Garlon 4® are as follows: "Dike large spills. Keep out of streams and domestic water supplies. Absorb small spills in inert materials such as sand." Recommendations for diquat by Chevron Chemical Company include directions to scrub the area using a detergent, after picking up the material with absorbants such as clay or loam soil.

Unused pesticide materials should be placed in leakproof containers, labeled, and transported by a licensed hauler to an approved hazardous waste disposal area. Unused pesticide material should not be flushed down drains. Glass, metal or plastic containers should be triple rinsed, crushed and placed in a waste storage drum, and taken to a local landfill. No pesticide container should ever be reused for any purpose, even after thorough washing. Additional information on container disposal can sometimes be found on the label, although some labels, directions, may not be recommended in Massachusetts (e.g., the Aminotriazole Weedkiller 90® label recommends that bags be burned).

Individuals should be consult the Massachusetts Pesticide Board for final recommendations. Additional detail is given in a document by the Armed Forces Pest Management Board (1980), from which much of this information was obtained.

CHAPTER 6. SOILS IN MASSACHUSETTS*

The following discussion presents some rough generalizations concerning the physical characteristics and terrain of 4 areas in the Commonwealth. The generalizations were obtained from a review of the Soil Conservation Service data sheets regarding the locations and descriptions of individual soils.

Western Massachusetts

In this area, typical soils are fine sandy loams (in which fine sand, 0.02 to 0.2 mm, is dominant) which frequently are overlain by thin organic matter. Bedrock outcroppings are common in these "rugged" areas, and depth to bedrock is often very shallow (less than 20 inches).

Slopes are as high as 50%-80%. The soils in this area are often well-drained (2.0 to 6.0 inches per hour) and often quite acidic (pH as low as 3.6). Lateral movement of water is likely where the bedrock is near the surface. Runoff can be expected to be rapid in areas of frequent bedrock outcropping

In areas where the depth to bedrock is greater, fine sandy loams are often underlain by compact glacial till. Frequently a fragipan, a densely compacted layer of soil, is found one to three feet below the surface. Above the fragipan, the soil is moderately permeable (0.6 to 6 in/hr), but once it reaches the fragipan it is slowed (to as a little as 0.06 in/hr), resulting in a tendency for the water to move laterally. Compared to the soils underlain by bedrock, these soils are less acidic (4.5 to 6.0) and rugged (slopes of 0 to 35%).

Central Massachusetts and Middlesex County

A variety of soil types exist in the area extending from the Connecticut River Valley to the coast although these soils are somewhat evenly

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*Information from Arthur D. Little, Inc. (1979).

distributed through this area. The main soil types found in the Boston area, for instance, are the same ones found near Worcester.

Some of the soils in this area are similar to those in Western Massachusetts, i.e., they are shallow fine sandy loams that are sometimes underlain by shallow bedrock and sometimes underlain by fragipans. Acidity and slopes are generally not as extreme in Western Massachusetts.

Other soils in this area are sandier ("loamy sands" instead of "sandy loams") and deeper (2-3 feet). These are well-drained soils (6 to 20 in/hr), especially in lower strata (greater than 20 inches per hour). Although slopes range from 0%-60%, runoff tends to be slow or moderate because of the permeability of the soil.

Also found in this area are deep muck soils that consist of decomposed organic matter (up to 50%) found in depressions and flat areas that are poorly drained (although the muck itself may be moderately permeable (0.6 to 6.0 in/hr). Occasionally, the muck is overlain by a sand layer (6.0 to 10 in/hr) which is deposited in these depressions from storm drainage from the surrounding sandy soils.

The soils in the central and Middlesex area (the shallow, fine sandy loams, the deeper sandier soils, and the muck) have pH values ranging from 3.6 to 6.5, with the most common values ranging from 4.5 to 6.0.

Southeastern Massachusetts

Soil types in Bristol and Plymouth counties are highly variable, with the most common being sandy loams. The range in common pH values is similar to other parts of the state (pH 3.6 to 6.0). The potential for water movement varies considerably:

- Rapid downward movement of water is likely in places where sandy loams overlie layers of increasing particle size for (i.e., where there is water-sorted stratification of sands and gravels).

- A more complex situation is found in places where a sandy loam with moderately rapid permeability (2.0 to 6.0 in/hr) overlies a fragipan layer that slows water movement to 0.2 to 0.6 in/hr. Just above the fragipan, there is a often layer of sand (permeability of 6 to 60 in/hr) which provides a duct for lateral movement of water which is blocked by the fragipan.

- In some places a fragipan can be found at a depth of 10 to 20 inches, and may be overlain for 7 to 9 months of the year by a perched water table.

- As described above, deep, moderately permeable muck soils are commonly overlain by a layer of sand that can encourage the lateral movement of water.

Cape Cod

Most of the rights-of-way on the Cape are in the western section (the "upper arm") of the Cape, where four soils are common:

- A loamy sand, overlying a gravelly coarse sand, which at a depth of about 27 inches, changes into very deep layers of gravel. The soil is rapidly permeable (6 to 20 in/hr) in the upper layers and extremely permeable (>20 in/hr) in the gravel substratum.

- A very deep coarse sand that is extremely permeable (>20 in/hr) from its surface to its substratum.

- A fine sandy loam, similar to those found in most other parts of the state, overlying a layer with more sand (a "loamy sand") and then a gravel soil. Permeability ranges from moderate (2 to 6 in/hr) to rapid (6 to 20 in/hr) from the surface to the substratum.

- A muck soil, similar to those discussed previously, found in concave areas.

Lateral movement of water is less likely on the Cape than in other places in Massachusetts, because of the propensity of water to move downward. Runoff may also be less of a problem for the same reason. The range of pH values is 3.6 to 6.0 on the Cape, as in many other parts of the Commonwealth.

CHAPTER 7. RARE PLANTS ON RIGHTS-OF-WAY

Method for Inventorying

In order to avoid destroying rare plants on rights-of-way, applicators must know the location of these plants. An inventory should be conducted to identify and map the rare plants. To inventory the rare plants found on existing rights-of-way, the following steps should be taken:

1. A trained field botanist should identify categories of habitat along the rights-of-way (i.e., wetlands, rocky slopes, calcareous areas) through the use of geological, soil, vegetation, and topographical maps, and field visits.
2. Plants should be collected in approximately 1/4 to 1/3 of each type of habitat. The ability to recognize differences between similar species (e.g., in the genus Cares) while collecting in the field is essential for to an accurate inventory.
3. After proper identification, mounted vouchers should be kept in a major herbarium (e.g., the New England Botanical Club, the University of Massachusetts, etc.), so that a permanent record of identities and localities is accessible to the interested public. Also, a list of plants collected should be published by the botanist in charge of the project.
4. Over the long term, an updated inventory can be maintained though encouragement of scientific and educational activity on rights-of-way. These activities, such as teaching several aspects of plant ecology and collecting, could generate additional information on new colonies of rare plants. Additionally, updated lists of rare plants should be obtained on a regular basis from the Massachusetts Natural Heritage Program (part of the Department of Environmental Management).

As shown in the following list, the endangered species are low-growing species. Selective spraying or cutting should minimize the effect on these species if care is taken to avoid contact with surrounding vegetation. To this effect, basal spraying may be less harmful than foliar spraying, and herbicides with narrow spectrums (particularly those

effective only on woody plants) would be less harmful than those with broad spectrums. The most important consideration may be the experience of the applicators in recognizing and avoiding known populations of rare plants.

Inventory of Rare Plants

The attached list of rare vascular plant species in Massachusetts was obtained from the Massachusetts Natural Heritage Program (Sorrie, 1983) and has been modified to include only those species likely to be found on existing rights-of-way. Therefore, the attached list does not include species exclusively found in habitats such as wooded areas, shore areas (tidal, brackish, sandy, or muddy) or saltmarshes. The decision to include or exclude species was based on published habitat information by Fernald (1950) and Coddington and Field (1978). Arrangement and nomenclature follow Kartesz and Kartesz (1980). Information on the habitat and substrate pH is indicated by number or letter codes as defined below:

1. Submerged in streams or ponds
2. Bogs, swamps, swales, or marshes
3. Peaty soil
4. River and stream banks
5. Lake or pond shores (sometimes in shallow water)
6. Meadows or grasslands
7. Shady rocks or ledges
8. Mountain slopes or summits
9. Sandy or gravelly soils
10. Dry exposed rocks or ledges
11. Roadsides, fields, or openings
12. Soils (not specified)

Substrate pH

a = acidic b = basic (i.e., calcareous)

Species for which habitat information was not available have been included in this list, although the likelihood of their being found on rights-of-ways is not known.

TABLE I-2

RARE PLANTS LIKELY TO BE FOUND ON RIGHTS-OF-WAY IN MASSACHUSETTS*

Species	Habitat**
EQUISETACEAE (Horsetails)	
Equisetum palustre Marsh Horsetail	c 3
E. variegatum Variegated Horsetail	3, 10
LYCOPODIACEAE (Clubmosses)	
Lycopodium alopecuroides Foxtail Clubmoss	2
L. carolinianum Carolina Clubmoss	3, 9
L. selago Fir Clubmoss	8
SELAGINELLACEAE (Spikemosses)	
Selaginella rupestris Rock Spikemoss	10
ISOETACEAE (Quillworts)	
Isoetes acadiensis Acadian Quillwort	-
I. eatonii Eaton's Quillwort	5
I. macrospora Lake Quillwort	5
OPHIOGLOSSACEAE (Adder's-tongue Ferns)	
Ophioglossum vulgatum Adder's-tongue Fern	6

*Adapted from Sorrie (1983).
**See text.

SCHIZAECEAE (Climbing and Curly Grass Ferns)	
Lygodium palmatum Climbing Fern	a 6
ADIANTACEAE (Cliff Ferns, Maidenhair Ferns)	
Cryptogramma stelleri Fragile Rock-brake	b 7
Pellaea atropurpurea Purple Cliff-brake	b 10
ASPLENIACEAE (True Ferns)	
Asplenium montanum Mountain Spleenwort	a 7, 12
A. ruta-muraria Wall-rue Spleenwort	b 10
SPARGANIACEAE (Bur-reeds)	
Sparganium minimum Small Bur-reed	4, 5
POTAMOGETONACEAE (Pondweeds)	
Potamogeton friesii Fries' Pondweed	b 4, 5
P. hillii Hill's Pondweed	5
P. strictifolius Straight-leaved Pondweed	b 5
ALISMATACEAE (Arrowheads, Water-plantains)	
Echinodorus parvulus Burhead	-
S. cuneata Wapato	4
S. teres Terete Arrowhead	4

POACEAE (Grasses)

Aristida purpurascens Purple Needlegrass	9
A. tuberculosa Seabeach Needlegrass	9
Calamagrostis pickeringii Reed-bentgrass	a 3, 6
Dichanthelium acuminatum var. wrightianum Wright's Panic-grass	5
D. ovale var. addisonii Commons's Panic-grass	9
D. scoparium Broom Panic-grass	9
Eragrostis frankii Frank's Love-grass	4
Muhlenbergia capillaris Hairgrass	10
Panicum gattingeri Gattinger's Panic-grass	11
P. philadelphicum Philadelphia Panic-grass	9, 11
Paspalum laeve var. circulare Paspalum Grass	6
Setaria geniculata Bristly Foxtail	9
Sporobolis heterolepis Northern Dropseed	10

CYPERACEAE (Sedges)

Carex alopecoidea Foxtail Sedge	2, 6
C. baileyi Bailey's Sedge	8
C. bushii Bush's Sedge	6
C. davisii Davis's Sedge	6
D. formosa Handsome Sedge	6
C. grayi Gray's Sedge	b 2
C. lenticularis Shore Sedge	4, 5
C. livida var. grayana Glaucous Sedge	2
C. michauxiana Michaux's Sedge	2
C. pauciflora Few-flowered Sedge	2
C. polymorpha	9

Variable Sedge	
C. schweinitzii	b 2
Schweinitz's Sedge	
C. sterilis	b 2
Dioecious Sedge	
C. tetanica	2, 6
Rigid Sedge	
C. trichocarpa	b 2, 4
Hairy-fruited Sedge	
C. typhina	b 2, 4
Cat-tail Sedge	
C. walteriana var. brevis	2
Walter's Sedge	
C. wiegandii	3
Wiegand's Sedge	
Cyperus engelmannii	2, 5
Engelmann's Umbrella-sedge	
Eleocharis equisetoides	5
Horsetail Spike-rush	
E. erythropoda	2, 5
Redfoot Spike-rush	
E. fallax	2, 5
Deceitful Spike-rush	
E. intermedia	b 5
Intermediate Spike-rush	
E. melanocarpa	5
Black-fruited Spike-rush	
E. obtusa var. ovata	2
Ovate Spike-rush	
E. quadrangulata	4, 5
Squarestem Spike-rush	
E. tricostata	2, 4, 5
Three-angled Spike-rush	
Psilocarya nitens	5
Short-beaked Bald-rush	
P. scirpoides	2, 5
Long-beaked Bald-rush	
Rhynchospora inundata	2, 5
Inundated Horned-rush	
R. torreyana	4, 5, 9
Torrey's Beak-rush	
Scirpus ancistrochaetus	2, 5
Barbed-bristle Bulrush	
S. hallii	5
Hall's Bulrush	
S. longii	2, 6
Long's Bulrush	
S. pendulus	2
Pendulous Bulrush	
Scleria pauciflora var. caroliniana	3, 9, 10
Papillose Nut-rush	
S. triglomerata	4, 5, 6
Tall Nut-rush	

ARACEAE (Arums)

Arisaema dracontium Green Dragon	2, 6
Orontium aquaticum Golden Club	2, 5

JUNCACEAE (Rushes)

Juncus biflorus Two-flowered Rush	5
J. pervetus Cape Cod Rush	2
Luzula parviflora ssp. melanocarpa Black-fruited Woodrush	8

SMILACACEAE (Catbriers)

Smilax bona-nox Bullbrier	9, 11

HAEMODORACEAE (Bloodworts, Redroots)

Lachnanthes caroliniana Redroot	2, 3, 9

IRIDACEAE (Irises)

Sisyrinchium arenicola Sandplain Blue-eyed Grass	6, 9

ORCHIDACEAE (Orchids)

Aplectrum hyemale Putty-root	8
Arethusa bulbosa Arethusa	2
C. reginae Showy Lady's Slipper	2
Listera cordata Heartleaf Twayblade	2
Malaxis brachypoda White Adder's Mouth	2
Platanthere ciliaris Orange Fringed Orchis	2
P. cristata Crested Fringed Orchis	2
P. dilatata Leafy White Orchis	2

P. flava var. herbiola Pale Green Orchis	2, 6
P. obtusata Bluntleaf Orchis	2
Spiranthes vernalis Grass-leaved Ladies' Tresses	6

SALICACEAE (Willows)

Salix candida Hoary Willow	2
S. exigua Sandbar Willow	4, 5
S. serissima Autumn Willow	b 2

BETULACEAE (Birches, Alders)

Alnus viridis ssp. crispa Mountain Alder	4
Betula pumila Dwarf Birch	2

FAGACEAE (Oaks, Beeches)

Quercus macrocarpa Mossy-cup Oak	b 6
Q. muhlenbergii Yellow Oak	b 12

POLYGONACEAE (Docks, Knotweeds)

Polygonum glaucum Seabeach Knotweed	9
P. puritanorum Pondshore Knotweed	4, 5
P. setaceum var. interjectum Strigose Knotweed	4, 5, 9

PORTULACACEAE (Purslanes, Spring-beauties)

Claytonia virginica Narrow-leaved Spring-beauty	2

CARYOPHYLLACEAE (Pinks, Sandworts)

Minuartia stricta Rock Sandwort	b 10

CARYOPHYLLACEAE (Pinks, Sandworts)

Minuartia stricta Rock Sandwort	b 10
Moehringia macrophylla Large-leaved Sandwort	b 4, 5, 9
Paronychia argyrocoma var. albimontana Silverling	10

NYMPHAEACEAE (Water-lilies)

Nymphaea tuberosa Tuberous Water-lily	4, 5

RANUNCULACEAE
(Buttercups, Crowfoots)

Clematis occidentalis Purple Clematis	b 10
Ranunculus circinatus var. subrigidus Stiff White Water-crowfoot	1
R. longirostris Beaked White Water-crowfoot	4, 5

MAGNOLIACEAE (Magnolias)

Magnolia virginiana Sweetbay Magnolia	2

PAPAVERACEAE (Poppies)

Adlumia fungosa Climging Fumitory	10

BRASSICACEAE (Mustards)

Arabis lyrata Lyre-leaved Rock-cress	9, 10
Cardamine douglassii Purple Cress	4, 5

PODOSTEMACEAE (Threadfoots)

Podostemum ceratophyllum Threadfoot	1

CRASSULACEAE (Sedums)

Crassula aquatica Pygmyweed	5

SAXIFRAGACEAE (Saxifrages, Currants)

Ribes americanum Wild Black Currant	2
R. triste Swamp Red Currant	2

ROSACEAE (Roses, Shadbushes)

Agrimonia pubescens Hairy Agrimonia	b, 7
Amelanchier bartramiana Bartram's Shadbush	8
A. nantucketensis Nantucket Shadbush	5
A. sanguinea Roundleaf Shadbush	b 12
Prunus pumila var. depressa Sandbar Cherry	4, 9
Rosa acicularis Prickly Rose	12
Sorbus decora Northern Mountain-ask	8, 11
Waldsteinia fragarioides Barren Strawberry	4, 5, 11

FABACEAE (Beans, Peas, Clovers)

Desmodium sessilifolium Sessile-leaved Tick-trefoil	9, 11
Lespedeza violacea Violet Bush-clover	11

OXALIDACEAE (Wood-sorrels)

Oxalis violacea Violet Wood-sorrel	9, 10

LINACEAE (Flaxes)

Linum intercursum Sandplain Flax	3, 4, 5, 9
L. sulcatum Grooved Flax	11

POLYGALACEAE (Milkworts)

Polygala senega Seneca Snakeroot	b 2

CALLITRICHACEAE (Water-starworts)

Callitriche anceps Northern Water-starwort	4, 5, 8
C. terrestris Terrestrial Starwort	11

EMPETRACEAE (Crowberries)

Corema conradii Broom Crowberry	9

CLUSIACEAE (St. John's-worts)

Hypericum adpressum Creeping St. John's-wort	3, 5, 9
H. stragulum St. Andrew's Cross	9

CISTACEAE (Rockroses)

Helianthemum dumosum Bushy Rockrose	9

VIOLACEAE (Violets)

Viola adunca Sand Violet	9
V. brittoniana Britton's Violet	3, 9

CACTACEAE (Cacti)

Opuntia humifusa Prickly Pear	9

LYTHRACEAE (Loosestrifes)

Cuphea viscosissima Blue Waxweek	11
Rotala ramosior Tooth-cup	5

MELASTOMATACEAE (Meadow Beauties)

Rhexia mariana Maryland Meadow Beauty	5

ONAGRACEAE (Evening-primroses)

Ludwigia polycarpa Many-fruited False-loosestrife	5
L. sphaerocarpa Round-fruited False-loosestrife	5, 9

HALORAGIDACEAE (Water-milfoils)

Myriophyllum alterniflorum Alternate-flowered Water-milfoil	1
M. pinnatum Pinnate Water-milfoil	5

APIACEAE (Parsleys, Angelicas)

Angelica villosa Hairy Angelica	11
Conioselinum chinense Hemlock Parsley	2
Hydrocotyle verticillata Water Pennywort	4, 5

ERICACEAE (Laurels, Blueberries)

Pyrola asarifolia var. purpurea Pink Pyrola	b 2
Rhododendron maximum Great Laurel	2

GENTIANACEAE (Gentians)

Sabatia campunulata Slender Marsh Pink	4, 5
S. kennedyana Plymouth Gentian	4, 5

ASCLEPIADACEAE (Milkweeds)

Asclepias verticillata Linear-leaved Milkweed	11

HYDROPHYLLACEAE (Waterleafs)

Hydrophyllum canadense Broad-leaved Waterleaf	4

BORAGINACEAE (Borages)

Onosmodium virginianum False Gromwell	9

VERBENACEAE (Vervains)

Verbena simplex Narrow-leaved Vervain	9

LAMIACEAE (Mints)

Agastache scrophulariifolia Purple Giant Hyssop	11
Blephilia ciliata Downy Wood Mint	11
B. hirsuta Hairy Wood Mint	6, 11
Scutellaria integrifolia Hyssop Skullcap	11
Trichostema brachiatum False Pennyroyal	b 12

SCROPHULARIACEAE (Figworts)

Agalinis acuta Sandplain Gerardia	9
Castilleja coccinea Indian Paintbrush	3, 6, 9
Mimulus alatus Winged Monkey-flower	5
M. moschatus Muskflower	2, 4
Pedicularis lanceolata Swamp Lousewort	2
Schwalbea americana Chaffseed	11

Veronica catenata Water Speedwell	b 12, 3
Veronicastrum virginicum Culver's-root	6

LENTIBULARIACEAE (Bladderworts)

Utricularia biflora Two-flowered Bladderwort	5
U. fibrosa Fibrous Bladderwort	5
U. subulata Subulate Bladderwort	3, 5, 9

RUBIACEAE (Bedstraws, Bluets)

Galium labradoricum Labrador Bedstraw	b 2
Hedyotis purpurea var. calycosa Calycose Bluet	10

CAPRIFOLIACEAE (Honeysuckles)

Lonicera hirsuta Hairy Honeysuckle	b 7
Symphoricarpos albus var. albus Snowberry	b 9
Viburnum rafinesquianum Downy Arrowwood	b 7

CAMPANULACEAE (Bluebells, Lobelias)

Lobelia siphilitica Great Blue Lobelia	2

ASTERACEAE (Asters, Composites)

Aster concolor Eastern Silvery Aster	9
A. prenanthoides Crooked-stem Aster	11
A. tradescantii Tradescant's Aster	10
Eupatorium leucolepis var. novae-angliae New England Boneset	3, 4, 5
Gnaphalium purpureum Purple Cudweed	9, 5
Petasites frigidus var. palmatus	2

Sweet Coltsfoot	
Sclerolepis uniflora	5
Sclerolepis	
Solidago hispida	4
Hispid Goldenrod	
S. macrophylla	8
Large-leaved Goldenrod	
S. ptarmicoides (=Aster p.)	b 9, 10
Upland White Aster	
S. rigida	b 10
Stiff Goldenrod	

APPENDIX II

INFORMATION ON INDIVIDUAL HERBICIDES

INTRODUCTION TO APPENDIX II

A number of people and organizations who commented on the report requested more information on the EPA registration and reregistration process, expressing concern that these chemicals are registered on the basis of unreliable or nonexistant data. The following discussion provides general information on the EPA process and highlights some of the issues which arise in evaluating the adequacy of that process. Additional information on the adequacy of the data base for each of the 14 herbicides has been added at the end of each of the chapters which follow.

The Federal Insecticide, Fungicide and Rodenticide Act (FIFRA), as amended, provides authority for a number of Federal activities, including the issuance of experimental permits, the conduct of research on biological controls, the certification of pesticide applicators, the issuance of permits for special local needs, and the exemption of certain pesticides from restriction in the case of an emergency. The most important part of the Act, however, directs EPA to decide what pesticides should be on the market, and what restrictions should be placed on those that are allowed to be marketed. Encompassed by this broad mandate are the specific sections in FIFRA devoted to registration, reregistration, suspension, cancellation, and decisions about tolerances (how much residue should be allowed on food), restricted vs. general use (how much training the applicator should have), toxicity category (how strong the warning label should be), reentry time (how much time should pass before farmworkers are allowed back on the field where the pesticide has been applied), and time-to-harvest (how soon after application can the crop be harvested).

Data which are considered in making these decisions include all of the types of data included in this appendix, plus usage data (crops, pests, application methods, seasonal timing, predicted geographic locations of usage); applicator exposure estimates and safety recommendations; recommended medical treatment in the case of emergencies; etc. Although the registrant is responsible for generating these data, EPA

can also accept data generated by other sources. EPA has access to all company data generated by the registrant in support of a registration. Acceptable protocols for generating data have been outlined by EPA.

To over-simplify the process of hazard evaluation, the data submitted by the registrant and other sources are compared to a set of criteria which attempt to define 'unreasonable adverse effect' (the "RPAR triggers"). Examples of these criteria include: a) a lack of an antidote, b) evidence of carcinogenicity, teratogenicity, or mutagenciity, and c) severe acute toxicity which protective measures cannot ameliorate. If these threshold criteria are exceeded, the registrant is given a chance to refute the conclusion that the pesticide causes an unreasonable adverse effect. The registrant, at this point, submits additional data which attempt to show that the adverse effect will not in fact occur, or that the benefits of the pesticide outweigh the risk of the adverse effect. If the registrant cannot show that either of these is true, the EPA can begin the process of cancelling the registration of the pesticide (or deciding not to register it in the first place). EPA then acts as an advocate in a judicial process, attempting to prove that the pesticide will have an unreasonable adverse effect, while the registrant attempts to prove the opposite, and the public is provided a chance to contribute data and opinions.

At the present time, the required data and the protocols outlined for generating these data are fairly extensive and thorough. However, this was not always the case. Most of the pesticides currently on the market were registered before the current registration requirements were in effect (the largest increase in data requirements took place in 1972). Many pesticides were in fact registered before EPA came into being in 1970; i.e., they were registered by USDA and 'grandfathered' into the set of products currently registered by EPA. In general, pesticides which were registered before 1972 do not have adequate data (many have no data) on a number of important topics, including carcinogenicity, teratogenicity, and mutagenicity. Even some registered after 1972 have inadequate data bases because acceptable protocols were not defined by EPA for several years. Thus, it is not possible to

state that a pesticide is "safe" because it is registered by EPA, since many of the pesticides currently on the market have not been reviewed by EPA, nor have they been tested to determine their potential hazard.

The obvious response to this situation is the suggestion that pesticides which have an inadequate information base should be removed from the market until data can be generated which show the pesticide to be safe; i.e., the general population should not be exposed as "guinea pigs" to chemicals of unknown safety. However, when Congress extensively amended FIFRA in 1972 and in subsequent years, no mechanism was given to EPA to remove a pesticide from the market because of inadequate data. To suspend or cancel a pesticide, EPA must show that the pesticide causes an unreasonable adverse effect. The burden of proof, in other words, is on the Agency to prove harm, rather than on the registrant to prove safety. (The only exception to this is that, once EPA officially requests information, the company has 90 days to relay its intention to provide those data. If the company does not respond in 90 days, EPA can move to suspend the pesticide. On the other hand, if the company responds within 90 days, there is no legal deadline for the subsequent submission of those data, and there is no way that EPA can remove the product from the market while the data are being generated.) Aside from the burden-of-proof problem, suspension of products with an inadequate data base poses another difficulty. Congress built in a strong disincentive for suspension decisions by stating that EPA must find the funds from within its own budget to remove the suspended product from the marketplace and dispose of it safely. Suspending the 35,000 pesticide products registered by USDA (prior to 1970) would therefore be financially impossible, even if EPA had statutory authority.

Recognizing the need to reexamine the products registered by USDA, Congress included in its 1972 amendments of FIFRA a requirement that EPA reregister these products by October 21, 1976. A number of activities, including a need to organize the old USDA files, prevented the reregistration effort from getting underway until 1975, when a few EPA people started going through the data on the 35,000 products.

At the same time, EPA was coming under pressure from Congress about the length of time necessary to conduct registration reviews. Single registrations on new products were taking years and many hundreds of man-hours to accomplish. Trying to meet the 1976 deadline, and knowing the resources required for a thorough review of data, a decision was made to concentrate on identifying and filling data gaps. As part of a Senate hearing on the matter in 1976, however, an independent toxicologist questioned the validity of the existing data in the old USDA files. In a report issued by the Senate Subcommittee on Administrative Practice and Procedure in December 1976, EPA was accused of negligence in its review of data for reregistration. EPA officials, meanwhile, were attempting to educate Congress, GAO, and its other accusers about the time and money necessary to review the 35,000 products with attention equally as thorough as that given new registrations. (At that time, EPA was registering less than 50 new pesticides a year, although the numbers varied considerably upon inclusion of different types of reviews, such as label changes, additional tolerances, etc.) In 1976 and 1977, EPA made requests for significant increases in staff and budget to handle reregistration. Finally, a small budgetary increase was provided for that purpose in 1978.

By this time, EPA had begun defining what is now known as the Registration Standards program. To make its task more manageable, EPA decided to concentrate not on the 35,000 separate products but on the 514 active ingredients in those products, with decisions on products grouped according to common active ingredients. According to the current approach, each standard attempts to:

1) Review the validity of the existing data and identify data gaps, which must be filled before the products can be reregistered.

2) Decide what products will be reregistered, and what restrictions will be imposed.

3) Decide whether the existing food tolerances of the product should be changed.

4) Decide what the label for each product should say.

This reregistration effort is now well underway, and is expected to be completed in 10 to 15 years at a cost of several hundred million dollars.

Meanwhile, questions about the validity of registration data were being raised by inspections of the laboratories generating those data. FDA discovered "serious deficiencies" (including fraudulent data) in a routine inspection of one of the laboratories of Industrial Bio-Test Laboratories, Inc. (IBT). In EPA, this discovery led to a moratorium placed on any registration applications which included data developed by IBT. In 1977, registrants were notified that they had to determine the validity of IBT tests according to EPA validation guidelines. The results of the registrant's evaluation (but not the raw data) would be reviewed by EPA, which would make the final determination of validity. After more problems were uncovered in an EPA/FDA inspection of two other IBT labs, EPA decided to request the raw data from the registrant so that EPA could conduct spot-checks of the registrants' audits. These spot-checks uncovered a number of problems in the audits of the registrants, and EPA and FDA (in conjunction with the Canadian government) decided to review each audit with the accompanying raw data.

The review of IBT studies was conducted between 1978 and 1983. It consisted primarily of 1) a validation review; i.e., do the raw data support the information in the final report, 2) an evaluation review; i.e., do valid studies conform to agency guidelines, and 3) a data gap review; i.e., is there another existing study in the chemical's data base to substitute for an invalid study. To save time, EPA eventually dropped the data gap review, assuming instead that an invalid IBT study had to be redone unless the registrant could come up with a substitute.

Midway through this review process, a number of decisions were made. The moratorium on registrations containing IBT data was lifted except for those registrations which depended on validation of an IBT study considered essential or critical to the evaluation. Thus, the normal review process could begin again on registrations which contained,

for example, an IBT-generated acute toxicity study, during the time that that IBT study was being validated or replaced. It was also decided that, if invalid IBT data comprised an entire data base, EPA could consider the product for cancellation. Finally, if it was discovered, in the replacement of an IBT test or by other input, that the pesticide had an adverse effect that had not been mentioned in the original application, EPA could expedite the regulatory process and initiate an intensive risk/benefit review or formal hearings on the status of the registration.

At the present time, the review of the IBT studies is nearly complete, and the remaining effort will be devoted to replacing the IBT studies (i.e., tracking the registrants' commitments to replace the studies). Of the 801 IBT test reviews, 74% were found to be invalid. A report written by EPA at the end of the review process concluded with the following comment:

> The IBT case caused serious concern and uncertainty about the potential hazards of the hundreds of pesticides involved, both for EPA and the public. Although it was advocated by some that all 212 pesticides tested in whole or in part by IBT be removed from the market pending retesting, that option is not available under current law. The regulatory response authorized by FIFRA requires valid evidence of risk, as opposed to a lack of information, before removing a product from the market, and allows for replacement of inadequate data. As we reach the final resolution of the IBT problem, it appears that this response was appropriate and adequate to deal with this event.

(By this, we can presume that EPA did not uncover a significant number of previously unknown adverse effects that would have warranted suspension of the products while the review was being conducted.)

Seven of the herbicides discussed in this report had registrations supported by some IBT data: atrazine, dicamba, diquat, glyphosate, metolachlor, picloram, and triclopyr. For each of these chemicals, Appendix II contains a brief discussion of the tests done by EPA and their status in regard to replacement. In general, most of the IBT studies have already been replaced. Dicamba remains as the one herbicide with a large number of data gaps still imposed by the IBT scandal.

In 1977, EPA and FDA established a joint audit program to make sure that another IBT situation has not already happened, and to make sure another does not happen in the future. Inspections are now made of the facilities, procedures, and staff qualifications of laboratories generating health effects data. Additionally, about 60 audits a year are conducted to see if the raw data concur with the reported results. According to EPA, "the large majority of laboratories" have been found to be acceptable, conducting scientifically valid tests in accordance with current standards. However, among the audits conducted between 1977 and 1980, 25 of the 82 laboratories audited were found to have serious deficiencies, including falsified research reports, contamination of feed with chemicals being investigated in other tests, and most frequently, improper record keeping.

The problem of public confidence in registration data has been further compounded over the last decade by an attempt by Monsanto and other large pesticide companies to limit public access to those data. These companies state that the public availability of registration data allows so-called "me-too" companies to register products after the patent runs out, or in foreign countries where U.S. patents do not apply. By making the data available to the public, Monsanto and others argue that the original registrant essentially subsidizes the other companies which want to make the same product. In response to these concerns, Congress has tightened the requirements for compensation by "me-too" companies for use of data submitted by the original registrant. However, Monsanto and others maintain that the problem still exists in regard to 'subsidizing' competitive registrations in foreign countries. There are some who believe that an additional reason for withholding data from the public is the fear that some environmentalists will search through the data looking for problems with which to challenge EPA's decisions.

The conflict over the availability of data also affects the review of data by the Commonwealth of Massachusetts. Companies want assurance that data submitted would remain confidential and only be examined by

qualified individuals. During extensive deliberations on this subject in the late 1970s, the Massachusetts Pest Control Board objected to these conditions, stating that the Commonwealth needed to provide information to a number of qualified people in the public, academic, and governmental sectors. Since the resolution of this question was pending in court, the Commonwealth decided not to set up procedures for protecting the proprietary nature of data submitted by companies. As described in the previous discussion of laws and regulation, the Supreme Court decided in June of 1984 to allow public disclosure of data provided in support of all FIFRA registrations, with certain limited exceptions (e.g., data on inert ingredients) similar to those exceptions originally included in the 1972 revisions of FIFRA.

* * *

The following discussion of herbicide data does not attempt to be a complete review of all available information. Because of time and budget constraints, only immediately available information was used. The contract for this work stated that secondary-source material (reviews of original studies) would provide the base of information for this report. Accordingly, a computer search of secondary-source material was undertaken and reports which were in the Boston area were identified and obtained. Reports which were considered to be particularly important were obtained by mail. The files of individual task force members were searched. Finally, requests for information were made of the companies which manufactured the 14 herbicides. The response of the manufacturers was uneven; however, the manufacturers which did respond sent both positive (i.e., showing no adverse effect) and negative data, although the former predominated.

Upon assembling the secondary-source material, each topic was reviewed to determine whether primary-source information (the original studies written by the investigator who conducted the work) was needed to supplement the information gathered so far. The following data bases were searched: BIOSIS, CHEM ABSTRACTS, AGRICOLA, MESH (Index Medicus, Medlars), and TOXLINE. Key studies were identified by: 1)

the attention given to them in the related secondary-source material, 2) their availability in the Boston area, and 3) the date of publication.

As can be seen in Appendix II, the adequacy of the data base varied considerably among the herbicides. Because many of the herbicides have such a poor data base, data generated by the manufacturer was included. This decision was criticized by several people who reviewed the draft GEIR, who felt that the incorporation of manufacturer's data jeopardizes the reliability of the conclusions drawn. Some felt that the manufacturer would be likely to falsify information in order to obtain a registration from EPA. The IBT scandal contributes to this fear. It is the authors' experience, however, that such falsification is the exception rather than the rule, and that most laboratories, whether company-owned or under contract, do not falsify data. Faced with evidence of adverse effect, most companies will not try to trick EPA into registering a product, if only because the financial risks are too high. The large initial costs of manufacturing and marketing the herbicide, plus the registration costs, would all be lost as soon as an independent researcher discovered the adverse effect and the RPAR mechanism was triggered.

Some reviewers also criticized the incorporation of company data because the information had not been peer-reviewed. However, the EPA registration process is as thorough as normal peer reviews. An IBT scandal does not disprove this since complete falsification of data is difficult to detect by any kind of peer review, as evidenced by the number of recent examples of complete data-falsification in the open literature.

A number of people who reviewed the draft EIR criticized the way that conclusions were drawn about the herbicides. Most of the criticism focused on the summary statements, contained in the main body of the report, regarding the toxicity of the herbicides. The concern was over statements regarding the lack of data which showed adverse effect. These reviewers felt that statements such as "available data showed no evidence of carcinogenicity" were misleading in light of the paucity of data available. These criticisms are valid, and a number of statements

have been revised to provide more emphasis on the lack of data and the uncertainty regarding potential adverse effects.

The suggestion was made by some reviewers to only consider studies which give positive results; i.e., which show adverse effect. The reasoning behind this suggestion is usually that:

1) The small statistical sample involved in any toxicity test makes a negative result less meaningful than a positive one.

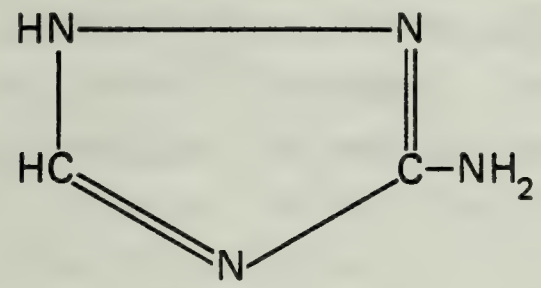
2) The lack of effect means only that the investigator did not see what he or she was looking for. Some effects, such as subtle or latent signs of biochemical or histological changes, need specially designed studies, and no tests are yet available for some effects, such as nausea, fatigue, headache, and minor central nervous system abnormalities.

Many chemicals, of course, do not cause adverse effects; negative data are the only way to show that this is true. The increased reliability of positive results, however, should cause the reader to question instances when there are only a few studies and these are negative. This is particularly true when the type of test is likely to produce false negative or false positive results.

A. AMINOTRIAZOLE

1. INTRODUCTION

Aminotriazole is the common name for the herbicide 3-amino-1,2,4-triazole from American Cyanamid Company (Thomson, 1975). It is also called Amitrole®, Amitrol®, ATA, Azolan®, Weed-Ar®, Weedazol®, Cytrole®, CytroAmitroleT®, Herbizole®, and Azole® (Thomson, 1975; TRW, 1981). Its chemical structure is



Aminotriazole is a stable, white crystalline solid (Carter, 1969). Other relevant physical and chemical characteristics are described below in the discussion of the fate of aminotriazole in soil and water.

2. TOXICITY

Acute Toxicity

Low acute toxicity values have been reported in a number of studies. Weir et al. (1958) reported an oral LD_{50} value of 14,700 mg/kg for mice and 25,000 mg/kg for rats. Washington State University and the U.S. Department of Agriculture (1971) assessed the toxicity of Amitrole-T, a formulation of amitrole that contains ammonium thiocyanate as an activator, and reported an LD_{50} value of 5000 mg/kg to rats. The same study reported a dermal LD_{50} value of greater than 10,000 mg/kg for rabbits. Norris (1976) stated that LD_{50} values ranged from 5000 to 25,000 mg/kg. Intravenous administration was studied by Weir et al. (1958), who reported that concentrations of 1600, 1750, and 1200 mg/kg had no effect on mice, cats, and dogs, respectively.

Intraperitoneal administration results in moderate toxicity of aminotriazole. The National Institute of Occupational Safety and Health, as cited in Lewis and Tatken (1982), reports that the intraperitoneal LD_{50} is 200 mg/kg.

Subacute/Subchronic Toxicity

Adverse effects have been reported in subchronic toxicity tests. In a study reported by Weir et al. (1958), rats were fed 1000 and 10,000 ppm for 63 days. Altered weight gain and fatty metamorphosis of the liver were observed. Jukes and Shaffer (1960) reported enlarged thyroid glands in rats fed 60 or 200 ppm aminotriazole for two weeks. The study associated this effect with a decrease in the uptake of radioactive iodine. Englehorn (1954) did not observe these changes at 15 or 30 ppm (no duration of exposure is given in a review by Durham and Williams, 1972).

Special Studies

Carcinogenicity Available data suggests that aminotriazole can be considered a carcinogen. Aminotriazole was used as a positive control in a carcinogenicity test by Innes et al. (1969). In that study, male and female mice (C57BL/6 × C3H/Anf and C57BL/6 × AKR) were fed 1000 mg/kg aminotriazole by gavage on days 7-28 of age. Thereafter, the mice were fed a dietary dose of 2192 ppm for 18 months. Carcinomas of the thyroid were found in 64/72 (89%) of the mice. In the C57BL/6 × C3H/Anf cross, hepatomas were found in 16/18 ((89%) of the males and 18/18 (100%) of the females, compared to 8/63 (13%) and 0/71 (0%), respectively, in untreated controls. In the C57BL/6 × AKR cross, 16/18 (89%) of the males and 17/18 (94%) of the females had hepatomas, compared to 4/72 (6%) and 1/65 (2%), respectively, in untreated controls.

Aminotriazole was used to confirm the ability of a new test system to detect carcinogens (Inoue et al., 1981). In this test, hamster embryo cell colonies were exposed in culture to aminotriazole for 8 days. Aminotriazole induced the expected morphological transformations at all

concentrations--10, 50, and 100 µg/ml. No such transformations were seen after exposure to phenyl salicate as the negative control.

Tsuda et al. (1976) found a statistically significant increase in invasive lesions and papillary adenoma nodules after administration of 2500 ppm aminotriazole in drinking water to Wistar rats. Invasion of follicular tissue through capsules into adjacent stromal tissue (considered to be evidence of malignancy) was observed in 19% of the treated rats, compared to 0% in the control group. Papillary adenoma nodules were found in 3% of treated rats, as contrasted with 0% in control groups.

As reported by an IARC Working Group (1974) that evaluated the carcinogenic risk of a number of industrial chemicals, several studies have indicated tumor induction by aminotriazole. In addition to the studies mentioned above, this report cites a study by Jukes and Shaffer (1960), who found dose-related induction of thyroid adenomas after administration of aminotriazole for 104 weeks at the relatively low levels of 10, 50, and 100 ppm. Thyroid adenomas developed in 1/10, 2/15, and 17/26 rats treated at the three dose levels. No tumors, except for a cystic follical, were found in control rats. Similar results were found by the Food Protection Committee (1959) as cited by Hodge et al. (1966). In that study, the same doses were administered (10, 50, and 100 ppm), and thyroid adenomas were observed in 1/27, 3/27, and 15/27 rats, respectively. In a third study (reported by the IARC Working Group), Napalkov (1969) found thyroid tumors in 7/22 male rats and liver tumors in 12/23 male rats (data on female rats were not provided) after administration of aminotriazole in drinking water at a rate of 20-25 mg/day/rat or in the food at 250 or 500 mg/day/rat.

A secondary source, Norris (1976), states that when rats were fed dietary concentrations of 50, 100, and 500 ppm aminotriazole for 476, 730, and 119 days, respectively, no adverse effects were seen at the lowest and highest doses, but that thyroid adenomas and adenocarcinomas were produced at 100 ppm. No primary source was provided. Another secondary source (USDOE, 1980) states that

"thyroid tumors began appearing in rats fed at 100 ppm for 68 weeks." No primary source was provided. The limited number of studies available on aminotriazole show no teratogenic effect, although more study is clearly needed.

Teratogenicity/Reproduction Aminotriazole does not appear to be a teratogen in mice. When aminotriazole was administered subcutaneously to mice at a level of 464 mg/kg (in 0.1 dimethyl sulfoxide) from day 6 to day 14 of gestation, no significant increase in malformations was seen among the offspring of treated mothers (USDHEW, 1969).

Aminotriazole has caused adverse effects in egg injection studies. Dunachie and Fletcher (1970) noted a 25% decrease in hatching at 100 ppm aminotriazole. Using high concentrations (20-40 mg/egg), Landauer and associates (1971) found up to 50% embryotoxicity after 96 hours of incubation. Increases in abnormalities, principally in formation of the beak, were noted at doses of 10 to 40 mg/egg. Treatment with doses of up to 2 mg/egg were without effect. A subsequent study (Landauer and Salam, 1972) found a slightly reduced incidence of terata when the aminotriazole was dissolved in dimethyl sulfoxide instead of water. The nature of the solvent, however, did not affect embryomortality, which was 60% with both solvents. The differing responses due to solvent are most likely attributable to solvent effects on cellular penetration and on distribution to sensitive sites.

It should be noted that chick egg studies are not considered meaningful in assessing risk to humans because of the absence of anatomical and physiologic maternal-fetal relationships during incubation.

Mutagenicity Although most of the studies shown in Table II-1 show no mutagenic activity of aminotriazole, the need for more study is indicated by the results of Kubinski et al. (1981), who found mutagenic activity when liver enzymes were added to a DNA cell-binding assay using E. coli.

Summary

An evaluation of important toxicity data is provided at the end of this chapter.

TABLE II-1

MUTAGENICITY TESTS: AMINOTRIAZOLE

Test System	Result	Comments	Source
Drosophila sex-linked recessive lethal	-	10 ppm	Laamanen et al. (1976)
	-	10 ppm	Sorsa and Gripenberg (1976)
Human lymphocytes in vitro	-	0.2-1%	Meretoja et al. (1976)
	-	<0.2%	Sorsa and Gripenberg (1976)
Host-mediated assay Mouse/Salmonella	-	1450 μ mole/kg orally plus equimolar dose sodium nitrite	Braun et al. (1977)
	+	2175 μ moles/kg as above	
Aspergillus nidulans			
Point mutation	-	2000 μg	Bignami et al. (1977)
Crossing over	(+)*	2000 μg	"
Nondisjunction	(+)*	2000 μg	"
Streptomyces coelicolor A3	-	1000 μg	Carere et al. (1978)
Bacillus subtilis "rec" assay	-		Shirasu et al. (1976)
Ames/Salmonella	-	5 μl	Anderson et al. (1972)
	-	0.4%	Bamford et al. (1976)
	-		Moriya et al. (1983)
	-		Carere et al. (1978)
	-		Shirasu et al. (1976)

TABLE II-1 (CONTINUED)

MUTAGENICITY TESTS: AMINOTRIAZOLE

Test System	Result	Comments	Source
Ames/Salmonella (continued)	-		Sorsa and Gripenberg (1976)
Escherichia coli WP2	- -		Moriya et al. (1983) Shirasu et al. (1976)
Escherichia coli pol A	- -	5 mg	Bamford et al. (1976) Sorsa and Gripenberg (1976)
Escherichia coli DNA cell-binding assay	+	10-100 μM plus liver enzymes	Kubinski et al. (1981)
rII mutants of T_4 bacteriophage	-	25 μg	Anderson et al. (1972)
Pelargonium zonale	+		Pohlheim and Gunther (1977)

* Parentheses indicate uncertainty.

3. MOBILITY AND PERSISTENCE

Fate in Soil

It is difficult to summarize the fate of aminotriazole, because its behavior appears to depend greatly on the nature of the soil. Although most chemicals show this variation with soil type, aminotriazole seems particularly variable in regard to both mobility and persistence. In general, aminotriazole can be expected to be mobile in sandy soils but immobile in organic ones or in soils with high clay content.

Kaufman et al. (1968) found that 40% of applied aminotriazole was adsorbed onto inorganic soils, while only 3% was adsorbed onto an inorganic soil. Leaching was found to occur readily in sand or sandy loam, but much less readily in clay, clay loam, or soil with a high organic content (Sheets, 1961). Marston et al. (1968) found that aminotriazole adsorbs readily and tightly to soils having a high base exchange capacity and a high percentage of organic matter. Sund (1956) also found adsorption to be dependent on the soil's base exchange capacity, and that aminotriazole may adsorb to soil colloids or complex with metallic ions (such as nickel, copper, iron, and magnesium).

Day et al. (1961), on the other hand, stated that adsorption was not a function of the base exchange capacity or the soil classification of 55 California soils. These authors conclude that aminotriazole would readily migrate since it is highly soluble in water, and the adsorbed aminotriazole can be released with sufficient infiltration of water. Specifically, Day and his colleagues found that although 20%-50% of the aminotriazole was adsorbed to a variety of soils, the bound aminotriazole could be released by repeated percolation of water through the soils.

Both volatilization and photodegradation are believed to be insignificant. The volatility of aminotriazole is stated as being low (no data provided), and the herbicide is considered to be stable under ultraviolet radiation. (Norris, 1971; Day et al., 1961; Ercegovich and Frear, 1965; Plimmer et al., 1967).

It is generally accepted that aminotriazole has a low to moderate persistence in soil, depending on soil type and conditions such as soil temperature and pH (TRW, 1981). Persistence increases with colder temperatures and decreases if the pH deviates above or below neutral (Ercegovich and Frear, 1965). The half-life of aminotriazole in a forest litter was found to be 5 days (Norris, 1970a). Burschel and Freed (no date provided) found a half-life of 6 weeks in a Chehalis loam soil and a breakdown rate of 1.31 µg/g soil/day. Freed and Furtick (1961) found that aminotriazole which had been applied at rates of 1-2 lb/acre on three Oregon soils could not be detected 2 months after application. After 6 days, recovery of aminotriazole from Hagerstown silt loam soil was 58%, 8%, and 0% from air-dried soil, soil with 15% moisture, and soil with 30% water, respectively (Gangstad, 1982; no primary source given).

It is uncertain whether the primary route of degradation is microbial or chemical. Limited success has been obtained in isolating soil microorganisms that degrade aminotriazole (TRW, 1981). The exhibition of a lag phase, typical of microbial degradation, indicates the possible importance of microorganisms. Additionally, it has been found that the degradation of aminotriazole almost stops if the soil is autoclaved (Day et al., 1961; Ercegovich and Frear, 1965; and Kaufman et al., 1968). Microbial breakdown may not be directly responsible for degradation, but may enhance or provide the conditions necessary for chemical degradation. Chemical degradation is suggested by the evolution of CO_2 from aminotriazole in the presence of free radicals (Plimmer et al., 1967; Kaufman et al., 1968). Whether the mechanism that opens the triazole ring is chemical or microbial, the resulting products (urea, cyanamide, and nitrogen) will be readily metabolized by microorganisms (Carter, 1975).

Persistence in Water

Available information suggests that aminotriazole does not persist in streams for more than a week (Norris et al., 1967; Marston et al., 1968). Norris (1967) showed that when aminotriazole was applied to 260 acres of a forested land at rates of 2 lb/acre, residual levels peaked within 2 hours and were undetectable by the third day. Residues of 422 ppb and 6 ppb were found after 0.17 hours and 8 hours,

respectively. In the study by Marston et al. (1968), 100 acres were treated with aminotriazole at a rate of 2 lb/acre. A maximum concentration (155 ppb) was attained 30 minutes after beginning application. After 2 hours, the aminotriazole concentration was 26 ppb; within 6 days the herbicide was no longer detectable.

It should be noted that these studies are difficult to interpret without information on dilution rates or herbicide sinks (degradation, accumulation in sediment, uptake by plants, etc.).

Indicators of Potential Ground Water Contamination

Table II-2 provides information on parameters associated with the mobility of aminotriazole. These parameters, and their associated thresholds, have been suggested by EPA for use in assessing the potential for pesticide contamination of ground water. A discussion of these parameters and thresholds, and the methods for arriving at designated values for individual herbicides, is presented in the main body of the report as part of the discussion of the fate of herbicides in the environment.

Toxicity Data Evaluation

Sufficient data exist to consider aminotrizole a carcinogen. However, available studies are insufficient to draw a conclusion about teratogenic effects. Additional studies regarding mutagenicity are needed because of the potential for activation by liver enzymes. At the time the draft GEIR was issued, a registration standard was being prepared by EPA to identify data gaps. However, that effort was terminated and a "special review" of aminotriazole was initiated in 1984, indicating a cause for concern had been found. The Scientific Advisory Panel, which guides EPA, was provided information on the chemical in June 1984. No further action has been taken by EPA at this date. The Commonwealth of Massachusetts is currently considering regulatory action regarding aminotriazole; a decision is forthcoming soon.

No data in support of the registration of aminotriazole were generated by IBT.

TABLE II-2

INDICATORS OF POTENTIAL GROUND WATER CONTAMINATION: AMINOTRIAZOLE

Indicator	Value for Aminotriazole	Threshold
Solubility	280,000 ppm at 23°C	>30 ppm
K_{oc}	0.059	<300-500
Speciation at pH 5	Neutral or cationic	Anionic (negatively charged)
Hydrolysis half-life	ND*	>6 months
Photolysis half-life	ND*	>3 days
Vapor pressure	Non-volatile	$<10^{-2}$ mm Hg

* ND = no data

4. TOXICITY TO NON-TARGET ORGANISMS

Birds

Aminotriazole appears to be non-toxic to test birds. Various studies by Heath et al. (1972), Hill et al. (1975), Maier-Bode (1973) and Pimental (1971) show LD_{50} values for mallard ducks to be greater than 2000 mg/kg, and LC_{50} values for mallard ducks, pheasants, Japanese quail, and ring-necked pheasants to be greater than 5,000 ppm in feed for 5 days. These data are summarized in Table II-3.

TABLE II-3

TOXICITY OF AMINOTRIAZOLE TO BIRDS

Species	Test	Result	Source
Mallard duck	LD_{50}	>2000 mg/kg	Maier-Bode (1973) Pimental (1971) Tucker and Crabtree (1970)
	5-day LC_{50}	>5000 ppm	Heath et al. (1972) Hill et al. (1972)
Japanese quail	5-day LC_{50}	>5000 ppm	Heath et al. (1972) Hill et al. (1975)
Pheasants	5-day LC_{50}	>5000 ppm	Heath et al. (1972)
Ring-necked pheasants	5-day LC_{50}	>5000 ppm	Hill et al. (1975)

Fish

Aminotriazole appears to be non-toxic to a variety of fish. A study by Bond et al., (1959) gave a 48-hour LC_{50} value for Coho salmon at 325 ppm. Hiltibran (1967) stated that bluegill, green sunfish, lake chubsuckers, and small-mouth bass fry were able to tolerate 50 ppm aminotriazole for 8 days with no observable adverse effects.

Lower Aquatic Organisms

An EPA report by Newton and Norgren (1977) said that Daphnia were "very sensitive" to aminotriazole, and the 48-hr LC_{50} value was given as 3 ppm. On the other hand, Crosby and Tucker (1966) cited the median immobilization concentration for D. magna as 23 ppm.

Bees

Aminotriazole may be slightly toxic to bees. The effect of dusting bees with 12.09 µg/bee produced a 5% mortality in a study by Atkins et al. (1973). Field doses of 1 kg/ha would equal 12 µg/bee.

Soil Microorganisms

A study by Fletcher (1960) concluded that when aminotriazole was applied at recommended rates of 8, 4, 2 and 1 lb/acre, the rate of nitrification was depressed and the number of microorganisms was reduced. A study by Ludzack and Mandia (1962) showed that aminotriazole inhibited microbial respiration and nitrification of activated sludge. Chandra (1964) showed a decrease of nitrifying bacteria in soil. Grossband and Wingfield (1978) showed that the decomposition of cellulose by fungi was reduced when high amounts (500 ppm) of aminotriazole were present in soil samples.

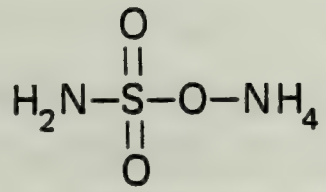
Bioaccumulation

Studies by Mullison (1979) and Newton (1979) stated that bioaccumulation was not of major concern because of aminotriazole's high solubility in water and its insolubility in cellular lipids. However, no data are available on the subject.

B. AMMATE

1. INTRODUCTION

Ammate® is the trade name for an inorganic herbicide, ammonium sulfamate, manufactured by E. I. du Pont de Nemours and Company. It is also called AMS, Amcide®, and Ammate X® (Thomson, 1977). It has the chemical structure shown below.



Relevant physical and chemical properties are presented below in the discussion of the fate of ammonium sulfamate in soil.

2. TOXICITY

Acute Toxicity

Acute oral toxicity studies show a low order of toxicity for ammonium sulfamate. The acute oral LD_{50} for ammonium sulfamate is 3900 mg/kg for rats (Du Pont, 1972b) and 5760 mg/kg for mice (Maki, 1973). When administered subcutaneously, the LD_{50} is 1438 mg/kg (Maki, 1973). No symptoms of irritation or systemic toxicity were noted from repeated applications of 20% or 50% aqueous solutions to the shaved skin of rats (Du Pont, 1972a). Likewise, no adverse reactions were observed in a test by Aoyama (1975) after ammonium sulfamate was administered to the skin of rats.

Subacute/Subchronic Toxicity

Du Pont (1972a) fed rats 10,000 ppm for 105 days and found no clinical signs of toxicity or pathology. Dietary administration of 100, 250, and

500 mg/l to rats for 90 days resulted in no effects on appearance, behavior, survival, or relative organ weights. At the highest dosage, body weights were reduced (Gupta et al., 1979).

Special Studies

There is very little information on the long-term toxicity of ammonium sulfamate. No indications of adverse toxicological effects (including effects on reproduction and lactation) were found by Sherman and Stula (1965) after administration of ammonium sulfamate in the diet of male and female rats for 19 months at dietary concentrations of 350 or 500 ppm. Although it is not clear whether tissues were examined microscopically for tumor induction, this study also suggests a lack of carcinogenic effect.

Negative results were noted for ammonium sulfamate (5 µl/plate) in an Ames/Salmonella assay (Anderson et al., 1972).

Summary

An evaluation of important toxicity data is provided at the end of this chapter.

3. MOBILITY AND PERSISTENCE

Fate in Soil and Water

Limited information is available on the environmental fate of ammonium sulfamate. Thomson (1975) states that ammonium sulfamate "breaks down rapidly in soil in the presence of moisture and high temperature." EPA (1981a) reviewed the data submitted for registration of ammonium sulfamate in a pesticide registration standard review in 1981. They stated that "the submitted data are insufficient to predict the fate of ammonium sulfamate (AMS) in the environment." The only study which EPA considered scientifically valid was done by Konnai et al. (1974), which showed that ammate is "very mobile" in the soil and exhibits a distribution parallel to mass flow. In this study, ammonium sulfamate (95% powder) was applied to an unspecified soil at high rates (50 kg/ha). After application of 2 cm and 50 cm of water, the ammonium sulfamate moved 14 cm and 50 cm, respectively.

Movement of ammonium sulfamate in the soil is suggested by its high water solubility, which is 216 g/100 ml at 25°C (Oullette and King, 1977) for the technical material. One formulation, AMMATE-X-N Weed and Brush Killer, has a somewhat lower, but still high, solubility of 68 g/100 ml water (Du Pont, 1972b). Ammonium sulfamate is considered non-volatile (Thomson, 1975, no data provided). At normal temperatures and pH, the hydrolysis rate is considered negligible (Du Pont, 1972a).

Indicators of Potential Ground Water Contamination

Table II-4 provides information on parameters associated with the mobility of ammate. These parameters, and their associated thresholds, have been suggested by EPA for use in assessing the potential for pesticide contamination of ground water. A discussion of these parameters and thresholds, and the methods for arriving at designated values for individual herbicides, is presented in the main body of the report as part of the discussion of the fate of herbicides in the environment.

TABLE II-4

INDICATORS OF POTENTIAL GROUND WATER CONTAMINATION: AMMATE

Indicator	Value for Ammate	Threshold
Solubility	2,160,000 ppm at 25°C	>30 ppm
K_{oc}	ND*	<300-500
Speciation at pH 5	Neutral (anionic and cationic charges cancel out)	Anionic (negatively charged)
Hydrolysis half-life	ND*	>6 months
Photolysis half-life	ND*	>3 days
Vapor pressure	Non-volatile	$<10^{-2}$ mm Hg

* ND = no data.

4. TOXICITY TO NON-TARGET ORGANISMS

Birds and Fish

Available data suggest that ammonium sulfamate is non-toxic to birds and fish. For quail, the LD_{50} is 3000 mg/kg (Maki, 1973). The 24-hr TL_m and 96-hr TL_m for channel catfish were found to be 259 ppm and 203 ppm, respectively, in a study by Clemens and Sneed (1959). A study by Alabaster (1969) found a range of 55 ppm to 3200 ppm for the LC_{50} for harlequin fish. Curtis and Ward (1981) found no significant mortality in fathead minnows when they were exposed to 600 mg/l of ammonium sulfamate for 96 hours.

Mammalian Wildlife

A study by Haugen (1953) indicated no signs of illness when deer were fed either crystals of ammonium sulfamate or foliage of various trees treated with ammonium sulfamate. The amount of herbicide consumed was not determined.

Soil Microorganisms

Thomson (1975) states that ammonium sulfamate "may cause temporary soil sterility." On the other hand, ammonium sulfamate was found to alleviate the inhibition of fungal growth caused by carbaryl (Cowley and Lichtenstein, 1970).

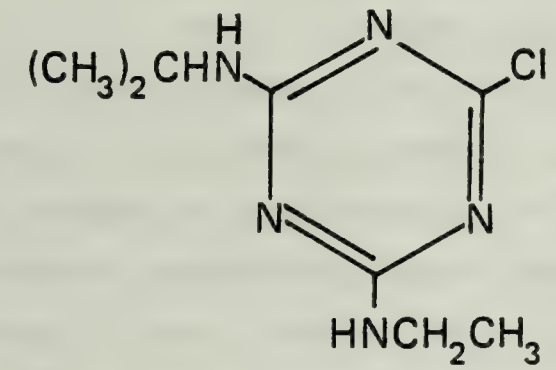
Toxicity Data Evaluation

Insufficient information is available to assess the carcinogenic, teratogenic, or mutagenic potential of ammonium sulfamate. EPA's registration standard pointed out these inadequacies as well as insufficiencies in the data regarding eye irritation, acute oral toxicity, acute dermal toxicity, subchronic oral toxicity, and subchronic dermal toxicity. No studies in EPA's registration files on ammonium sulfamate were conducted by IBT.

C. ATRAZINE

1. INTRODUCTION

Atrazine is the common name of the herbicide 2-chloro-4-ethylamino-6-isopropylamino-s-triazine. It is produced by Ciba-Geigy and is marketed under the trade name AAtrex. Combinations of atrazine with other herbicides include Bicep 4.5L® (atrazine and metolachlor), Atratol 80W® (atrazine and prometon), Atratol 8P® (atrazine, sodium chlorate, and sodium metaborate), and AAtram 80G® (atrazine and propachlor). The structure of atrazine is:



Relevant physical and chemical properties are described below in the discussion of the fate of atrazine in soil and water.

2. TOXICITY

Acute Toxicity

Acute oral toxicity studies show a low order of toxicity for atrazine. Oral LD_{50} values, summarized in Table II-5 range from 750 mg/kg to 3080 mg/kg for rats, mice, rabbits, and hamsters. Lewis and Tatken (1980) reports a dermal LD_{50} value of 7500 mg/kg. When 38 mg of atrazine is applied to the skin of a rabbit, a mild irritation results. Application of 6.32 mg to rabbit eyes (standard test for eye irritation) results in a severe reaction.

TABLE II-5

ACUTE ORAL TOXICITY OF ATRAZINE

	(LD_{50}, mg/kg)				
Form	Rats	Mice	Rabbits	Hamsters	Source
Atrazine	3080				Thomson (1975)
	3080				Ouellette and King (1977)
	3800	1750			Mullison (1977)
	1750	1750	750	1000	Lewis and Tatken (1982)

Subacute Oral Toxicity

On the basis of a limited number of reports, atrazine does not cause any observable adverse effects when fed to cattle, dogs, and horses. In one subacute toxicity study, atrazine in an 80% wettable powder form was fed to cows at either 100 ppm for 21 days or 30 ppm for 4 weeks. In each case, no ill effects were observed (Ciba-Geigy, 1971). One report states that in studies in which 25 ppm atrazine was fed to dogs, cattle, and horses for extended periods of time, no ill effects were observed (Mullison, 1979).

Mammalian Metabolism

In rats given radiolabeled atrazine, 85% was excreted in urine and feces after 72 hours. The remaining radioactivity was found in the lung, liver, and kidney, with lower concentrations in muscle and fat. The most common metabolic reactions are dealkylation of the amino group, hydrolysis of the 2-chloro-group, and oxidation of an N-alkyl side-chain to carboxylic acid and alcohol (Erickson et al., 1979).

Special Studies

Carcinogenicity Only one study was available regarding the carcinogenicity of atrazine. This study suggests that atrazine is not carcinogenic in mice. Male and female mice (C57BL/6 × C3H/Anf) and (C57BL/6 × AKR) were given 21.5 mg/kg by gavage from days 7 to 28 of age, followed by a dietary dosage of 82 ppm for approximately 18

months. No increase in incidence of tumors above control values was noted (Innes et al., 1969).

Teratogenicity/Reproduction No teratogenic effects were observed in a study by the U.S. Department of Health, Education, and Welfare (1969). Atrazine was administered subcutaneously to mice at a level of 46.4 mg/kg (in 0.1 ml dimethyl sulfoxide) from days 6 to 14 of gestation. No significant increase in malformations was observed among the offspring of treated mothers.

Reproduction In a series of experiments, Peters and Cook (1973) examined the effects of atrazine on reproduction in rats. No effect on the number of pups/litter or weaning weight was seen in the offspring of rats fed up to 1000 ppm atrazine in the diet throughout gestation. Subcutaneous injections of up to 200 mg atrazine/kg of days 3, 6, and 9 of gestation had no effect on the number of pups per litter. No indications are provided on whether any other parameters were monitored.

Subcutaneous injections of higher doses (800-2000 mg/kg) under the same test conditions were embryotoxic. One of the seven rats injected with 1000 mg atrazine/kg had a normal litter; all embryos were resorbed in the six other dams in this treatment group and in the group given 2000 mg/kg. One of four dams in the 800 mg/kg groups had no pups. (It is unclear from the data presented whether the authors looked for resorptions and found none, or did not monitor this effect in this group.) Although the authors did not report maternal toxicity, it appears highly likely that maternal toxicity did occur above 800 mg/kg. Atrazine is moderately toxic to rats when administered by injection. The lowest reported lethal dose by injection (intraperitoneal) for this species is 550 mg/kg (Lewis and Tatken, 1982). The embryotoxicity noted at levels above 800 mg/kg may therefore be an indirect response to toxic effects in the dams.

Using the egg injection technique, Dunachie and Fletcher (1970) noted a decrease (27%) in hatching in chick eggs injected with 400 ppm atrazine

(in 90% methanol). No effect was observed at 300 ppm. It should be noted that a great deal of variability in the percentage hatching was observed, and no clear dose response was evident for any of the tested herbicides.

Mutagenicity As shown in Table II-6, most of the available studies indicate that atrazine is not mutagenic. The data suggest that activation by plant enzymes is required to produce a mutagenic response, and that mammalian liver enzymes appear incapable of activation.

Other Information Bontoyan et al. (1979) screened a variety of technical and commercial pesticide formulations for the presence of nitrosamine contaminants. Negative findings were reported for atrazine. The limit of detection, however, was only 1 ppm.

Summary An evaluation of important toxicity data is provided at the end of this chapter.

3. MOBILITY AND PERSISTENCE

Fate in Soil

Atrazine is generally considered to have limited mobility (Newton and Norgren, 1977; Schlapfer, 1977; Kozlowski and Kuntz, 1973), and it is readily adsorbed onto soil particles (Von Rumker et al., 1975; Witt and Baumgartner, 1979). Koslowski and Kuntz (1973) report that leaching of atrazine is also insignificant in sand, based upon a study of atrazine which was applied to Plainfield sand at rates of 1, 2, 4 lb/acre. When 2, 4, and 8 inches of water were used, most of the atrazine remained in the first inch of soil. The application of additional water resulted in the leaching of a portion of the atrazine downward to a depth of 6 inches. Similar results were obtained by Marriage et al. (1975). When atrazine was applied to plots of sandy loam soil at a rate of 4.5 kg/ha for 9 consecutive years, the herbicide remained in the upper 15 cm of soil, and the majority of that was in the 0-5 cm soil layer. The maximum residue level was measured as 0.4 kg/ha in the top 15 cm of soil. In a loam soil to which 2-20 lb a.i./acre atrazine was applied and which received 8.16 inches of rainfall, 85.3% of the applied atrazine was found in the top 1 inch and 5.7% was found in the 1-2 inch layer, after one year (Birk and Roadhouse, 1962).

TABLE II-6

MUTAGENICITY TESTS: ATRAZINE

Test System	Result	Comments	Source
Mouse dominant lethal	-		Chollet et al. (1982)
In vivo mammalian cytogenetics			
Mouse spermatogonia	-	1 ppm in drinking water for 7 weeks	Chollet et al. (1982)
Mouse bone marrow	-	1 ppm in drinking water for 7 weeks	"
Mouse spermatogonia	-	6 mg/kg i.p.	"
Mouse bone marrow	-	6 mg/kg i.p.	"
Mouse bone marrow	-	2 g/kg + liver enzymes	Zaccaro et al. (1980)
Mouse bone marrow	+	2 g/kg + plant enzymes	"
Drosophila dominant lethal	+	AAtrex 80W® (80% atrazine)	Murnik and Nash (1977)
Drosophila sex-linked recessive lethals	-	10 mM	Adler (1980)
	-		Gopalan and Njagi (1981)
	+	Larval feeding with 0.01%	Murnik and Nash (1977)
In vitro mammalian cytogenetics			
Chinese hamster ovary cells	-	10 mM	Adler (1980)
Chinese hamster V79 cells	(+)	3 mM	"
	-	Liver enzymes added	Zaccaro et al. (1980)
	+	Plant enzymes added	"
Ehrlich ascites tumor cells	(+)	10^{-4} M for 3 hr	Walker et al. (1979)

TABLE II-6 (CONTINUED)

MUTAGENICITY TESTS: ATRAZINE

Test System	Result	Comments	Source
Host-mediated assay			
Mouse/*E. coli*	+	100-600 mg/kg orally	Adler (1980)
	+	Orally	Solt and Neale (1980)
Mouse/*Salmonella*	-	1000 mg/kg	Adler (1980)
Mouse/yeast	-	1 g/kg + liver enzymes	Zaccaro et al. (1980)
	+	1 g/kg + plant enzymes	"
Aspergillus nidulans	(+)	Plant enzymes added	Adler (1980)
	-	8000 ppm for 4 hrs	deBertoldi et al. (1980)
Streptomyces coelicolor	(+)	Plant enzymes added	Adler (1980)
Schizosaccharomyces pombe	(+)	6 mM; plant enzymes added	Adler (1980)
	+	Plant enzymes added	Zaccaro et al. (1980)
	-	Liver enzymes added	"
	-	3 mM	Chollet et al. (1982)
Saccharomyces cerevisiae	-	10 mM; plant enzymes added	Adler (1980)

TABLE II-6 (CONTINUED)

MUTAGENICITY TESTS: ATRAZINE

Test System	Result	Comments	Source
Saccharomyces cerevisiae DW mitotic conversion	-	Liver enzymes added	Zaccaro et al. (1980)
	-	Plant enzymes added	"
	-	4000 ppm for 1 hr	de Bertoldi et al. (1980)
	-	1000 ppm for 16 hrs	"
	+	Water soluble extract of maize plants exposed to 10-25 ppm atrazine	Plewa and Gentile (1976)
Ames/Salmonella	-	100 µg/plate + liver enzymes	Lusby et al. (1979)
	-		Shirasu et al. (1976)
	-	Liver enzymes added	Adler (1980)
	-		Anderson et al. (1972)
	-	Liver enzymes added	Zaccaro et al. (1980)
	-	Plant enzymes added	"
	+	Point mutations without liver enzymes	Gopalan and Njagi (1981)
	+	Water-soluble extract of maize grown in atrazine-treated soil (strain TA100; same results seen with plant grown in untreated soil)	Bakshi et al. (1981)
Bacillus subtilis "Rec" assay	-		Shirasu et al. (1976)

TABLE II-6 (CONTINUED)

MUTAGENICITY TESTS: ATRAZINE

Test System	Result	Comments	Source
Escherichia coli	- -		Shirasu et al. (1976) Adler et al. (1980)
rII mutants of T_4 bacteriophage	-	20 µg	Anderson et al. (1972)
Pelargonium zonale	+		Pohlheim and Gunther (197

Other studies have shown higher mobility. In one study in which atrazine was applied at concentrations of 0.5-2 ppm to a Sharpsburg silty clay loam soil, atrazine residues (amount not given) moved to a depth of 90 cm (ca. 36 inches) and remained there for after 41 months (Lavy et al., 1963). Residues at shallower depths dissipated during that time. Similar results were found by Harris et al. (1970), who found that atrazine moved to a depth of 38 cm (15 inches) and persisted longer at these lower depths. Atrazine was applied to three sandy loam soils in Nevada and Idaho at rates of 1.2 to 9.6 lb a.i./acre atrazine. After 1 year, residues in the top 6 inches of soil ranged from 0.005 ppm to 0.25 ppm. In the next 6 inches of soil (6-12 inches below the surface) residues ranged from 0.05 ppm to 0.15 ppm (EPA, no publication date provided [a]).

A number of factors have been found to influence the mobility of atrazine by influencing the amount of atrazine that is adsorbed. Adsorption is greater at lower temperatures, at lower pH, under dryer conditions, and, especially, in soils with higher percentages of organic matter (TRW, 1981).

Several field studies have been done to determine the potential for surface water contamination from runoff containing atrazine. Von Rumker et al. (1975) suggests that the potential for high concentrations of atrazine in runoff water is significant, especially if rainfall occurs in the first 2 weeks after application.

Triplett et al. (1978) studied the runoff after atrazine had been applied to several watersheds ranging from 0.4 to 3.5 ha in area. The highest concentration of atrazine detected was 0.48 ppm, which occurred soon after application.

Atrazine is generally considered a persistent herbicide. A review article by Sheets (1970) suggests that in agricultural soils, residues often persist at phytotoxic levels for greater than 1 year. Several studies (Buchanon and Hiltbald, 1973; Ritter et al., 1974; and Best et al., 1975) suggest that atrazine can persist 1 to 2 years. The data

presented above regarding the mobility of atrazine substantiates its persistence, since in all of the studies samples were taken after a year or more. One study mentioned above in regard to mobility suggests a particularly long persistence. In a loam soil that received 2-20 a.i./lb acre atrazine and 8.16 inches of rainfall, a total of 91% of the applied atrazine remained after 1 year in the top 2 inches of soil.

Although the amount of loss by volatilization is not fully understood, this route of loss is generally considered to be insignificant because of atrazine's low vapor pressure (ranging from 5.7×10^{-8} at 10°C, to 2.3×10^{-5} at 50°C) (TRW, 1981). Volatilization of atrazine occurs generally in the first 2 days after application (Mullison, 1979; Hammons, 1977). Loss by photodecomposition can be significant if residues are subjected to high temperatures and prolonged sunlight before precipitation. Photodecomposition would probably not be significant, therefore, on shaded soil (TRW, 1981).

Although atrazine is considered to be a stable compound, chemical and microbial degradation are known to occur and have been studied extensively. Chemical degradation of atrazine by a first-order hydrolysis reaction has been observed to occur in soil (Zimdahl, 1970; Armstrong et al., 1967). The rate of hydrolysis is a function of pH and is base and acid catalyzed such that at pH values of 2.2, 3.1, 11.1, and 11.9, the half-lives of atrazine are 18.4, 66.4, 81.1 and 15.2 days, respectively.

Two studies (EPA, no publication date provided [a]; Kearney et al., 1977) proposed that nitrosoatrazines may be formed if $NaNO_2$ is present in concentrations of 100 ppm (as N) or greater and at pH values of 3 or less. However, since these conditions rarely occur, the formation of such compounds should be insignificant in natural soil systems.

Microbial decomposition occurs by three routes, dealkylation, ring cleavage, and the hydroxylation of the 2-chloro group (TRW, 1981). Dealkylation is believed to be the primary mechanism and was observed to occur in the presence of 12 different types of fungi, 2 of which were

Rhizopus stolonifer and Aspergillus fumigatus (Kaufman and Blake, 1970). Ring-cleavage reactions were studied by Roeth et al. (1969) and by McCormick and Hiltbold (1966). Both showed that microbial cultures degraded the atrazine to hydroxy-atrazine by a ring cleavage. Further degradation of the hydroxy-atrazine was found to be three times faster than degradation of the parent compound in a mixed microbial population. These secondary reations give rise to metabolites which may be subject to further degradation (Kearney and Kaufman, 1975; Ramsteiner et al., 1972).

Fate in Water

There have been very few studies concerned with the fate of atrazine in water. Klaasen and Kadoum (1979) studied the distribution and decay of atrazine that had been applied at an initial concentration of 0.3 ppm to a farm pond. The atrazine appeared to be persistent in the water and the mud. The concentrations measured at days 1 and 120 decreased from 309 to 206 ppb in the water and from 323 to 204 ppb in the mud. In a 1975 study (EPA, no publication date provided [a]) of seven major rivers in the central United States, peak residues occurred during the months of heaviest application (May to June). After June, the residual levels were ≤ 1 ppb. The maximum concentration of atrazine detected was 16.7 ppb.

Photodecomposition of atrazine probably does not occur to any significant extent in water, due to its extremely slow reaction rate (Wolfe et al., 1976). It has been shown in the laboratory that photolysis of aqueous atrazine yields 2-hydroxy-atrazine. Laboratory photolysis of aqueous atrazine with ultraviolet radiation at 253.7 nm yields a 2-hydroxy compound. This is probably due to the nucleophilic displacement of the chlorine atom (Wolfe et al., 1976).

Indicators of Potential Ground Water Contamination

Table II-7 provides information on parameters associated with the mobility of atrazine. These parameters, and their associated thresholds, have been suggested by EPA for use in assessing the potential for pesticide contamination of ground water. A discussion of these

parameters and thresholds, and the methods for arriving at designated values for individual herbicides, is presented in the main body of the report as part of the discussion of the fate of herbicides in the environment.

TABLE II-7

INDICATORS OF POTENTIAL GROUND WATER CONTAMINATION: ATRAZINE

Indicator	Value for Atrazine	Threshold
Solubility	33 ppm at 27°C	>30 ppm
K_{oc}	81.6	<300-500
Speciation at pH 5	Neutral or catonic	Anionic (negatively charged)
Hydrolysis half-life	42 days (pH 5)	>6 months
Photolysis half-life	>14 days (natural light)	>3 days
Vapor pressure	3.0×10^{-7} mm Hg at 20°C	$<10^{-2}$ mm Hg

4. TOXICITY TO NON-TARGET ORGANISMS

Birds

Atrazine appears to be non-toxic to test birds. Heath et al. (1972) and Hill et al. (1975) reported LC_{50} values from a 5-day diet to be greater than 5000 ppm for bobwhite quail, Japanese quail, ring-necked pheasants, and mallard ducks. Tucker and Crabtree (1970) reported an LD_{50} for mallard ducks to be greater than 2000 mg/kg. Studies by Mullison (1979) and by Heath et al. (1972) reported LD_{50} values for mallards and pheasants to be greater than 5000 ppm, and for bobwhite quail, 700-800 ppm.

Fish

Available data suggest that atrazine is toxic to some species of fish. Toxicity data are summarized in Table II-8. Studies by Mullison (1979), and by Newton and Norgren (1977) showed that goldfish and bluegill sunfish had 48-hr LC_{50} values of 118 mg/l; Butler (1965) showed that rainbow trout had a 48-hr LC_{50} of 4.5 mg/l. The authors concluded that atrazine had a low toxicity to goldfish and bluegill, but was toxic to rainbow trout.

Vivier and Nisbet (1965) used atrazine in the form of A361 and found that 0.5 ppm of A361 was lethal to 20% of a minnow population in 72 hours. The TL_m for minnows was 1.25 ppm. When atrazine in the form of Gasaprime was used, the 24-hr and 48-hr TL_m values for minnow were 3.75 and 2.5 ppm respectively. Jones (1962) found a survival rate of 90% for 72-hour exposures for Micropterus salmoides fry at 5.0 ppm, Letalurus puctatus at 10.0 ppm, and Lepomis macrochirus at 10.0 ppm. A review study by EPA (no publication date given [a]) noted a 20%-30% reduction in the growth of a variety of fishes (including gizzard shad, channel catfish, bluegill sunfish) when treated with 500 µg of atrazine per liter of water. The number of offspring was reduced by 96% at both 20 and 500 µg/l.

Lower Aquatic Organisms

In field studies by Walker (1964), atrazine was applied to ponds at concentrations of 0.2 to 6.0 ppm. The author concluded that atrazine was somewhat toxic to bottom fauna. Mayflies, caddis flies, leeches, and gastropods were among the most sensitive species. The bottom fauna appeared to recover in 4 to 6 months after treatment. Walker (1962) applied 0.5-2.0 ppm of atrazine to ponds and found that clams were reduced to 1/8 of their original number, while the snail population increased four-fold. Fingernail clams, isopods, and damselflies showed no mortality when subjected to 20 µg/l and 500 µg/l of atrazine in an EPA study (publication date not given [a]). Additional data is summarized in Table II-9.

TABLE II-8

TOXICITY OF ATRAZINE TO FISH

Formulation	Species	Test	Result (ppm)	Source
A 361	Minnow	48-hr TL_m	1.25	Vivier and Nisbett (1965)
Gasparime	Minnow	24-hr TL_m	3.75	"
	Minnow	48-hr TL_m	2.5	"
Atrazine	Bluegill	48-hr LC_{50}	118	Mullison (1979) Newton and Norgen (1977)
Atrazine	Goldfish	48-hr LC_{50}	118	Mullison (1979) Newton and Norgen (1977)
Atrazine	Rainbow trout	LC_{50}	4.5	Butler (1965)
Atrazine	Spotfish	48-hr LC_{50}	No effect at 1.0 ppm	"

Indirect Effects on Aquatic Ecosystems

A review study by EPA (publication date not given [a]) found that small ponds exposed to small amounts (20 and 150 µg/l) of atrazine for 135 days showed an immediate decline in the rate of photosynthesis by aquatic algae. Within a few weeks, the growth of atrazine-resistant plant species increased. Zooplankton reproduction rates (Simocephalus and Daphnia) were reduced by 57% and 70% respectively with 500 µg/l, and by 9% and 70% respectively with 20 µg/l. By day 15, the zooplankton biomass was reduced by as much as 60% with both 20 µg/l and 500 µg/l, and the species composition was affected by both concentrations of atrazine.

Effect on Livestock

Mullison (1979) reported that cattle, dogs, and horses fed a diet of 25 ppm atrazine over an extended period of time produced no observable adverse effects. Palmer and Radeleff (1969) reported the toxic dosage of atrazine for cattle to be 25 mg/kg after 8 doses by drench and 2 doses by capsule. Chickens given 10 doses at 50 mg/kg had significant reductions in weight gain. The toxic dosage for sheep was 5 mg/kg, although it appeared that some sheep may be less sensitive than the average.

Bees

No data was found on the effect of atrazine by itself on bees. However one study by Sonnet (1979) that studied the synergistic effect of atrazine with insecticides showed no significant mortality when bees were fed sublethal doses of insecticides plus atrazine, as compared to being fed only insecticides.

Bioaccumulation

The limited data available suggests that atrazine may concentrate to a limited extent in a number of organisms. In data provided by the EPA (publication date not given [a]) maximum residues in fish were estimated at 500 ppb when the concentration in the water was 16.7 ppb. At a near-normal concentration of 1.0 ppb in water, the concentration in fish was 20 ppb. After treatment of a model aquatic ecosystem with

TABLE II-9

TOXICITY OF ATRAZINE TO LOWER AQUATIC ANIMALS

Species	Test	Result	Source
Shore crab	LC_{50}	>100 ppm	Portman and Wilson (1971)
Cockle	"	>100 ppm	"
Brown shrimp	"	10-30 ppm	"
Oyster	96-hr EC_{50}	No effect at 1.0 ppm	Butler (1965)
Shrimp	48-hr EC_{50}	30% at 1.0 ppm	"

0.82 ppm atrazine (in bottom soil), Kearney et al. (1977) found bioaccumulation ratios in algae, fish, and snails to be 9, 16, and 8, respectively. Percich and Lockwood (1978) have done a study indicating that atrazine accumulates in some species of fungi.

Soil Microorganisms

Studies of atrazine's effect on soil microorganisms seem to produce varying results. A study by Percich and Lockwood (1978) showed atrazine to be a growth stimulant to microflora when Conover loam soil was treated with 10, 30, and 100mg/g. Studies done in the Soviet Union by Kozlova et al. (1967), Milkowska and Gorzelak (1966), and Sosnovskaya and Pashchenko (1965) confirmed these data.

In comparative studies done by Volts et al. (1974), it was shown that atrazine applied at a rate of 4 kg/ha reduced populations of anaerobic bacteria, sporeformers, cellulolytic microorganisms, and nitrifying, amylolytic, and denitrifying microbial groups.

Toxicity Data Evaluation

Insufficient information is available regarding the carcinogenicity and teratogenicity of atrazine. In regard to carcinogenicity, relevant tests have recently been submitted by the manufacturer to EPA for review, and EPA has reviewed one of these studies. Although no statement has been issued by EPA, it can be reasonably assumed that this test did

not show positive effect, since no "rebuttable presumption against registration" resulted from this review. A similar statement can be made for the rodent teratogenicity test, which has been recently submitted to and accepted by EPA.

While it is never possible to say that no further study is needed, the data base regarding mutugenicity for atrazine is sufficient to draw the conclusion stated several paragraphs above. The testing of mutagenic potential is an inexact science--some false positive or false negative studies are to be expected. In regard to atrazine, only eight of the 47 studies conducted show positive findings without activation by plant enzymes. Given the extensive amount of study this subject has already received, a judgement must be made on the basis of the majority of results. In regard to the possibility of activation independent of the liver, mentioned by the Conservation Law Foundation in its review of the draft GEIR, our review concluded that insufficient data were provided by Adler (1980) to justify his suggestion of this possibility.

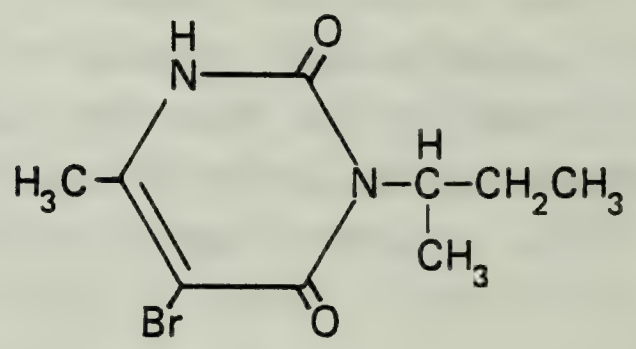
The registration standard for atrazine cites the need for a non-rodent teratogenicity study, a gene mutation, and a gene metabolism study, and an update of the reproduction study currently on file in order to satisfy recent protocol guidelines. (Other deficiencies which are not related to mammalian toxicology include data on hydrolysis, photodegradation in soil, metabolism in soil and water, leaching potential, soil degradation, and accumulation in crops, fish, and lower aquatic organisms.)

Two chronic oral studies in EPA registration files were conducted by IBT. Portions of both of them were found to be valid, and EPA has decided both can be used for supplemental information. One study has been replaced by Ciba-Geigy; no response has yet been made regarding the replacement of the other study. Other chronic oral studies on atrazine in EPA files have been conducted by other laboratories.

D. BROMACIL

1. INTRODUCTION

Bromacil is the common name for the herbicide 5-bromo-3-sec-butyl-6-methyl uracil, produced by E. I. du Pont de Nemours and Company. Its formulations are known as Hyvar®, Hyvar-XL®, Hyvar- XP®, Nalkil®, Urox B®, Urox-HX®, Ureabor®, Boracil®, Borea®, Hibor®, Instemul®, Bro-40®, and Uragen® (Thomson, 1975; Ouellette and King, 1977; EPA 1980a). Bromacil is a substituted uracil compound with the following structure:



Relevant physical and chemical parameters are presented below in the discussion of the fate of bromacil in soil and water.

2. TOXICITY

Acute Toxicity

Bromacil appears to have a low acute toxicity. Ouellette and King (1977) and Thomson (1975) list the oral LD_{50} value of bromacil to laboratory animals as 5200 mg/kg. A technical data sheet reports the acute oral LD_{50} for dogs to be greater than 5000 mg/kg. With dermal application, the acute lethal dose was found to be greater than 5000 mg/l (maximum feasible dose). No toxic symptoms were observed at this dose (Du Pont, 1979a). The inhalation LC_{50} for rats is greater than 4.8 mg/l, using an 80% formulation (Du Pont, 1979a).

Bromacil appears to be moderately irritating to the skin when applied as a 50% aqueous suspension (80% WP) to intact or abraded skin of guinea pigs. No skin sensitization occurred. A slight transient conjuctival irritation of rabbit eyes occurred after administration of 10 mg of dry 50% powder, or 0.1 ml of a 10% suspension in mineral oil. No corneal injury occurred (Du Pont, 1979a).

Mammalian Metabolism

Gardiner (1975) states that substituted uracil herbicides can be expected to be excreted rapidly by animals (no data was provided for bromacil). Two principal urinary excretion products were found to be 5-bromo-3-sec-butyl-6-hydroxymethyl uracil and 5-bromo-3-(2-hydroxy--1-methylpropyl)-6-methyl uracil.

Special Studies

Carcinogenicity No signs of carcinogenicity were seen in 2-years chronic studies with rats and dogs (Sherman and Kaplan, 1975; data were not included in publication). Charles River-CD rats (36 of each sex) were fed 0, 50, 250, and 1250 ppm bromacil in a diet supplemented by 1% corn oil. Dogs (1-2 year old beagles, 3 of each sex) were fed the same dosages, with levels gradually increasing throughout the study.

Teratogenicity and Reproduction Sherman and Kaplan (1975; data not included in the report) found no gross manifestations of a teratogenic effect or abnormalities in bone structure when primagravid rabbits were fed 0, 50, and 250 ppm bromacil on days 8-16 of gestation, with offspring delivered on day 29 or 30 by Caesarian section or normal parturition. No results were reported on fetal weights or number of resorptions, although these data were apparently obtained.

Newell and Dilley (1978, unpublished data) exposed 50 Sprague-Dawley rats to vapors/aerosols of bromacil (particle size range of 0.3 to 3.0 μm) for 1 to 3 hours on days 7 to 14 of gestation. Concentrations of bromacil in the air were 0, 38, 78, and 165 mg/m^3. At the highest dose (165 mg/m^3) a slightly higher percentage of resorptions was

found, compared to controls. The study also found dose-related reductions in fetal weight and caudal ossification (significant at $p < 0.01$). No effects were noted on weight gain, food consumption, average number of pregnancies, or litter size. No terata or signs of gross pathology were noted.

Sherman and Kaplan (1975, data not included in publication) conducted a rat reproduction study in which Charles River-DC rats (12 of each sex) were exposed to 0 and 250 ppm bromacil for three generations. In each generation, no effects were noted on the number of matings, pregnancies, or offspring in each litter (at birth and at 4, 12, and 21 days). No effects were noted in the body weights of offspring at 21 days. Gross and microscopic examination of third-generation pups revealed no abnormalities.

Other Chronic Effects Sherman and Kaplan (1975; data not included in publication) observed a follicular cell adenoma and a slightly higher incidence of focal light cell hyperplasia and focal follicular hyperplasia in the thyroids of rats receiving 1250 ppm bromacil in a diet supplemented by 1% corn oil. No abnormalities in hematology, biochemistry, or urinalysis were noted. Tibia length and organ weights were not affected at any dosage. In females, at the highest dosage (1250 ppm) a slight decrease in food consumption and weight gain was noted (significant at $p < 0.001$).

In a 2-year dog study by the same authors, beagles (1 to 2 years old, 3 of each sex) were exposed to gradually increasing doses of bromacil in their diet, with in final dosage levels of 0, 50, 250, and 1250 ppm. No compound-related changes were noted in hematology, biochemistry, urinalysis, or pathology. At 1250 ppm in both sexes, an initial slight decline in body weight was followed by a stabilization.

Mutagencity As shown in Table II-10, most available studies indicate that bromacil is not a mutagen. Of particular note is the negative result obtained by Epstein et al. (1972) using a high dose of 1000

TABLE II-10

MUTAGENICITY TESTS: BROMACIL

Test System	Result	Comments	Source
Mouse dominant lethal	-	Single i.p. injection of of 150 mg/kg	Epstein et al. (1972)
	-	Five p.o. administrations of 1000 mg/kg	"
	-	Diet for 7 weeks; doses not given	Jorgenson et al. (1976, abstract)
Drosophila dominant lethal	(+)*	Response appears to toxicity-related rather than an induction of chromosomal breaks or loss	Murnik (1976, abstract)
Drosophila sex-linked recessive lethal	-		Gopalan and Njagi (1976, abstract)
Saccharomyces cerevisiae D3 Mitotic recombination	-		Simmons et al. (1976, abstract)
	-		Riccio et al. (1981, abstract)
Saccharomyces cerevisiae D7 Mitotic crossing-over and gene conversion			Riccio et al. (1981, abstract)

TABLE II-10 (CONTINUED)

MUTAGENICITY TESTS: BROMACIL

Test System	Result	Comments	Source
Bacillus subtilis rec assay	-		Shirasu et al. (1976)
Ames/Salmonella	-		Shirasu et al. (1976)
	-		Simmons et al. (1976, abstract)
	-		Moirya et al. (1983)
	-	5 µl/plate	Anderson, et al. (1972)
	+	point mutations without liver activation	Gopalan and Njagi (1976, abstract)
Escherichia coli WP2	-		Noriya et al. (1983)
	-		Shirasu et al. (1976)
	-		Simmons et al. (1976, abstract)
rII mutants of T_4 Bacteriophage	-	50 µg/plate	Anderson, et al. (1972)
Unscheduled DNA synthesis (human fibroblasts)	-		Simmons et al. (1976, abstract)

* Parentheses indicate uncertainty.

mg/kg in a mouse dominant lethal study, considered one of the more reliable tests of mutagenic potential.

Summary An evaluation of important toxicity data is provided at the end of this chapter.

3. MOBILITY AND PERSISTENCE

Fate in Soil

A review of registration material led EPA to conclude that "Bromacil is highly mobile in soil, leaching to depths of 18 to 24 inches" (EPA 1980a). Leistra et al. (1975) found high mobility when bromacil was applied annually for 6 to 7 years at rates of 1.6 and 2.4 kg a.i./ha to a sandy loam and a silty clay loam. (In the sandy loam, the organic matter percentages were 2.3, 1.9, 1.4, and 0.5% in the 0-20, 20-40, 40-60, and 60-100 cm layers, respectively. In the silty clay loam, the organic matter percentages were 3.1, 2.5, 1.5, and 0.6.) One year after final application, residues of 0.01-0.02 µg/g of bromacil were found down to the 80-100 cm layer in sandy loam and the 50-60 cm layer in silty clay loam. Highest concentrations (16-33 µg/g) were found in the 10-20 cm layer in the sandy loam.

Smith et al. (1975) found a similar high mobility in an unspecified soil in irrigation ditches. Bromacil leached to a depth of 90 cm over a period of 3 years. When the ditch initially filled with water, the authors found lower concentrations of bromacil than the other herbicides tested. They attributed this to the high solubility of bromacil and its tendency to leach out of the upper zones of soil. Bromacil is soluble to water at 815 ppm at 25°C (Ouellette and King, 1977).

Reed (1982) found different mobility patterns for bromacil, using three soils with varying percentages of organic matter. Bromacil was applied at a rate of 8.96 kg/ha to a sandy loam with 0.56% organic matter, a silty clay loam with 1.52% organic matter, and a sandy loam with 2.48% organic matter. Figures II-1 and II-2 show the movement of bromacil at 6 weeks and 6 months respectively. In the soil with the lowest percentage of organic matter, with limited retention capacity, bromacil apparently moved through the soil, and was found in very limited

Concentrations of Bromacil in Soil

Figure 1

6–8 Weeks after Treatment

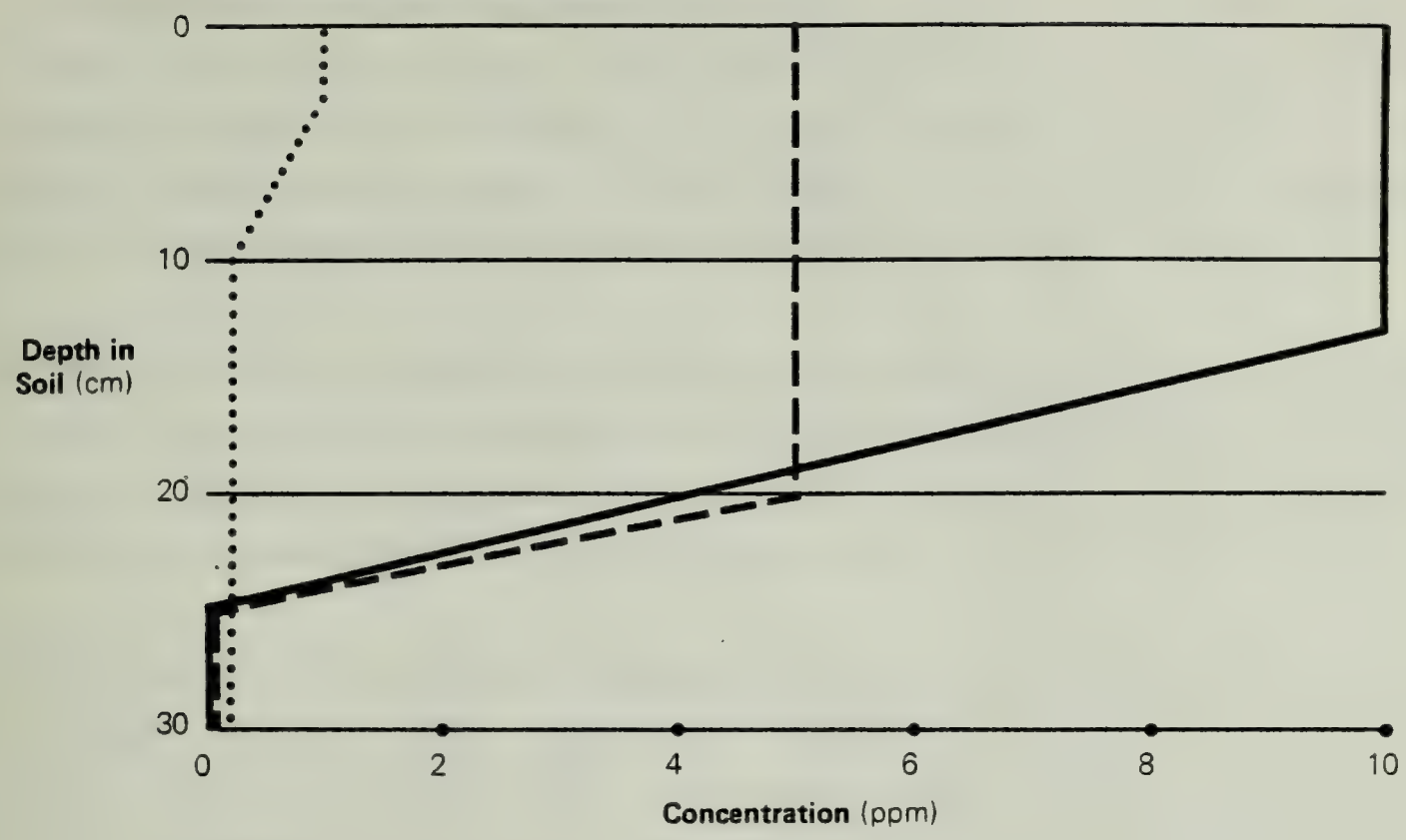
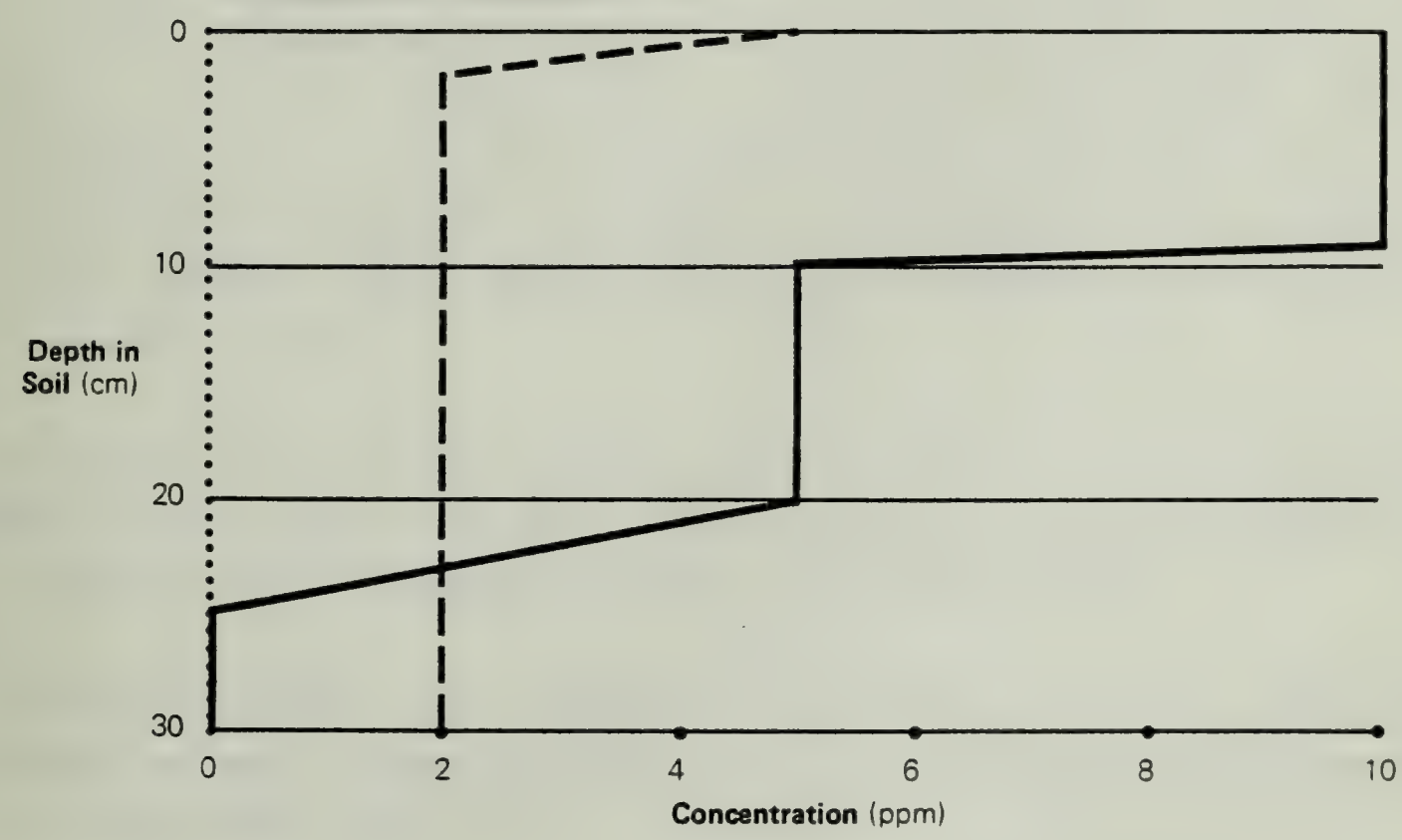


Figure 2

23 Weeks after Treatment



Soil with low organic matter

Soil with moderate organic matter

Soil with high organic matter

Adapted from Reed 1982

quantities throughout the 30 cm soil profile. None remained after 6 months. In the soil with intermediate amounts of organic matter, approximately 5 ppm was found throughout most of the profile after 6 weeks, dropping to about 2 ppm after 6 months. In the soil with the highest organic matter content, 10 ppm bromacil was retained in the upper layers after 6 weeks. After 6 months, concentrations increased in the lower depths while remaining high in the upper layers.

Helling (1970) also found the mobility of bromacil to depend on organic matter. According to Helling and Turner's mobility classification system, bromacil is in class 3 to 5 (with 5 representing the greatest mobility) when tested in soils with high to low organic matter content.

Bromacil can be considered persistent in soil. In a review of its registration material, EPA found the half-life in soil to be 7 months (unspecified soil) (EPA 1980a). In a sandy loam used by Leistra et al. (1975), discussed above, the rate of decrease in concentration corresponded to a half-life of 8 months. A somewhat shorter persistence was found by Gardiner et al. (1969), who determined the half-life in a silt loam to be 5 to 6 months, and by Jolliffe et al. (1967), who found half-lives of 3 to 6 months in unspecified California soils. When radioactive-labeled bromacil was applied to unspecified soil in the field, 68.8% remained after 5 weeks, 63% after 14 weeks, and 23.5% after 1 year (EPA, 1980a).

Microbial activity is the primary mechanism of breakdown of bromacil in the soil (Torgeson and Mee, 1967). The major metabolic product is 5-bromo-6-hydroxymethyl-3-sec-butyluracil (EPA, 1980a).

Photodecomposition and volatilization are not expected to be significant routes of loss. Losses of bromacil from soil were found to be less than 0.1% per week (Hill, 1971).

Persistence in Water

Only limited information is available on the fate of bromacil in water. Bromacil is not easily photolyzed, but does form 5-bromo-6-methyluracil

at a very low rate. Degradation by hydrolysis does not appear to be significant (EPA, 1980a).

Indicators of Potential Ground Water Contamination

Table II-11 provides information on parameters associated with the mobility of bromacil. These parameters, and their associated thresholds, have been suggested by EPA for use in assessing the potential for pesticide contamination of ground water. A discussion of these parameters and thresholds, and the methods for arriving at designated values for individual herbicides, is presented in the main body of the report as part of the discussion of the fate of herbicides in the environment.

4. TOXICITY TO NON-TARGET ORGANISMS

Birds and Fish

Thomson (1975) and a study by EPA (1975a) state that bromacil is non-toxic to birds and fish. The 8-day dietary LC_{50} was found to be >10,000 ppm for both mallard ducklings and bobwhite quail (Du Pont, 1979a). The 48-hr LC_{50} values for bluegills and carp are 71 ppm and 164 ppm, respectively. The 96-hr LC_{50} for fathead minnows is 182 ppm. Rainbow trout show greater sensitivity, with a 72-hr LC_{50} of 28 ppm (Du Pont, 1979a).

Lower Aquatic Organisms

Limited information suggests that bromacil is non-toxic to lower aquatic organisms. The 3-hr TL_m values for crayfish and water fleas are both >40 ppm. The 72-hr TL_m for crayfish is 230 ppm.

Bees

Atkins et al. (1976) classifies bromacil as "relatively non-toxic" to honey bees.

TABLE II-11

INDICATORS OF POTENTIAL GROUND WATER CONTAMINATION: BROMACIL

Indicator	Value for Bromacil	Threshold
Solubility	815 ppm at 25°C	>30 ppm
K_{oc}	72	<300-500
Speciation at pH 5	ND* (probably neutral due to 2 weakly basic and no acidic groups)	Anionic (negatively charged)
Hydrolysis half-life	Appears to be stable (EPA, 1980a)	>6 months
Photolysis half-life	Appears to be stable (EPA, 1980a)	>3 days
Vapor pressure	2.5×10^{-7} mm Hg at 25°C	$<10^{-2}$ mm Hg

* ND = no data.

Toxicity Data Evaluation

Insufficient information is publicly available on the carcinogenicity and teratogenicity of bromacil. One carcinogenicity study has been accepted by EPA. It is reasonable to assume that this test did not show positive results, since no rebuttable presumption against registration was triggered. Another carcinogenicity test is required by the registration standard. Other data requirements yet to be filled by the manufacturer include a chronic feeding study, and one reproductive effects study. (Other deficiencies include data on acute toxicity to estuarine and marine organisms, avian single-dose toxicity, acute fish toxicity, acute toxicity to aquatic invertabrates, hydrolysis, photodegradation in water, metabolism in soil and aquatic organisms, leaching potential, dissipation, and accumulation in crops, fish, and lower aquatic organisms.) No further mutagenicity tests are required. Again, it can be assumed that the mutagenicity tests on file do not show bromacil to be a mutagen, since no further regulatory action has been taken. The majority of

tests shown in Table II-10, including a mouse dominant lethal study, strongly suggest that bromacil is not a mutagen.

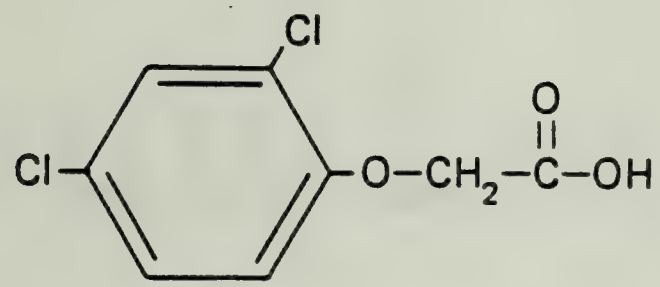
Regarding teratogenicity, the data cited above are consistent in the indication of no teratogenic effect. One additional test, however, is needed to satisfy EPA requirements. One teratogenicity test has already been accepted by EPA.

No data in EPA registration files concerning bromacil were generated by IBT.

E. 2,4-D

1. INTRODUCTION

2,4-D is the common name for the herbicide 2,4-dichlorophenoxy acetic acid, available from Dow Chemical U.S.A. and others. It is produced in numerous formulations, some of which are Weedone®, LV-4®, Esteron 99® Concentrate, Weedar 64®, DMA-4, Verton 2-D®, Agrotect®, Barwell®, Phenox®, Weed-B-Gon®, Miracle®, and Formula 40®. (TRW, 1981; EPA 1980b). These formulations involve a variety of forms of 2,4-D, including sodium salts, amines, high volatile esters, low volatile esters, and oil-soluble mixtures (Thomson, 1975). The structure of the acid is:



More than other herbicides, the physical and chemical properties of 2,4-D are dependent on the form of the active ingredient. Table II-12 shows some of the variation in solubility for various forms of 2,4-D. Other physical and chemical properties are presented below in the discussion of the fate of 2,4-D in soil and water.

2. TOXICITY

Acute Toxicity

In a report by NRCC (1978), 2,4-D was considered moderately toxic, based on the oral LD_{50} values summarized in Table II-13. The oral LD_{50} values for the acid form range from 100 mg/kg in the dog to 541 mg/kg in chicks. Both Drill and Hiratzka (1953) and Rowe and Hymas (1954) noted that the salts and esters were less toxic than the acid form.

TABLE II-12

VARIATIONS IN THE SOLUBILITY OF 2,4-D

Form	Solubility in Water
Acid	0.09g/100g @ 25°C
Diethylamine salt	300g/100g @ 20°C
Butoxyethanol ester	Insoluble
N-oleyl-1,3 propylene-diamine salt	Insoluble
DMA-4® (dimethylamine salt)*	Infinite
Esteron-99® Concentrate*	Emulsifiable
Formula 40®	Infinite

* As cited by Dow Chemical U.S.A. (1978, 1980); all others as cited by TRW (1981).

The Material Safety Data Sheet for Weedar 64® (the DMA salt of 2,4-D acid) by Union Carbide (1977) cited the oral LD_{50} value for male albino rats as 1615 ± 170 mg/kg for the salt formulation. This sheet reported the dermal LD_{50} value as greater than 500 mg/kg, and the inhalation LD_{50} as greater than 288.6 mg/l, both for the rat.

The Material Safety Data Sheet for Weedar 64® stated that it was an eye irritant to male rabbits. The label for DMA-4 by Dow Chemical U.S.A. states, "Warning. Injurious to eyes, may cause skin irritation." The Material Safety Data Sheet for the same substance reported that "eye contact may cause moderate irritation and also moderate corneal burn. Skin contact may cause moderate irritation and possibly a superficial burn." Approximately the same conclusions were drawn on the Material Safety Data Sheets for both Esteron 99® and Formula 40® (Dow Chemical U.S.A., 1980ab).

TABLE II-13

ACUTE TOXICITY OF 2,4-D

Formulation	Species	Sex	LD_{50} (mg/kg)	Source
Acid	Mouse	M	368	Rowe and Hymas (1954)
	Rat	M	375	"
	Rat	-	<500	McLaughlin (1951)
	Guinea pig	-	<320	"
	Guinea pig	M,F	469	Rowe and Hymas (1954)
	Dog	M,F	100	Drill and Hiratzka (1953)
	Chicks	M,F	541	Rowe and Hymas (1954)
Alkanolamine	Chicks	M,F	>380,<765	Bjorn and Northern (1948)
Amino Salt	Chickens	F	1950	Loktionov et al. (1973)
	Rat	-	1200	"
	Mouse	-	300	"
Butoxy ethyl ester	Chicks	M,F	900	Whitehead and Pettigrew (1972a)

TABLE II-13 (CONTINUED)

ACUTE TOXICITY OF 2,4-D

Formulation	Species	Sex	LD_{50} (mg/kg)	Source
Butyl esters	Mouse	F	713	Rowe and Hymas (1954)
	Rat	F	620	"
	Guinea pig	F	848	"
	Rabbit	F	424	"
	Rat	-	920-1500 in water 300-400 in diesel fuel	Stupnikov (1972) "
	Mouse	-	380-640 in water	"
Isopropyl ester	Rat	M,F	700	Rowe and Hymas (1954)
	Mouse	M	541	"
	Guinea pig	M	550	"
Potassium salt or sodium salt	Rat	M	>100	Bjorklund and Erne (1966)
Sodium salt	Mouse	-	375	Hill and Carlisle (1947)
	Mouse	-	360	Lotionov et al. (1973)
	Rat	F	805	Rowe and Hymas (1954)
	Rat	-	666	Hill and Carlisle (1947)
	Guinea pig	M	551	Rowe and Hymas (1954)
	Guinea pig	-	1000	Hill and Carlisle (1947)
	Rabbit	-	800	"
	Monkey	-	>214	"
	Rat	-	730	Loktionov et al. (1973)

TABLE II-13 (CONTINUED)

ACUTE TOXICITY OF 2,4-D

Formulation	Species	Sex	LD_{50} (mg/kg)	Source
PGBE esters	Rat	F	570	Rowe and Humas (1954)
DMA-4 (6.3% dimethyl-amine salt	Guinea pig	-	820	Dow Chemical U.S.A Material Safety Data Sheet (1977)
	Rabbit	-	710	"
	Rat	F	1230	"
	Rat	M	1070	"
Formula 40® (56.5% alkanolamine salt)	Rat	-	1000-2000	Dow Chemical U.S.A. Material Safety Data Sheet (1977)
Esteron 99® (68.6% 2,4-D, propylene glycol butyl ether esters	Rat	F	760	"

Subchronic Toxicity

Drill and Hiratzka (1953) reported that dogs could tolerate 10 mg/kg/day of 2,4-D, 5 times per day, for 13 weeks without any significant adverse effects. The dogs could not tolerate 20 mg/kg for the same time period (adverse effects not stated in the 1978 NRCC review). Row and Hymas (1954) gave oral doses of 3, 10, 30, 100, and 300 mg/kg of 2,4-D to rats, 5 times per week for 4 weeks. No observable effects were noted at concentrations below 30 mg/kg, but at 30 and above, a depressed growth rate, liver pathology, and gastrointestinal irritation were noted. Rats given 300 mg/kg died within the 4-week period.

Mammalian Metabolism and Elimination

Khanna and Fang (1966) fed 1 to 100 mg of ^{14}C-labeled 2,4-D to rats and monitored expiratory gases, urine, feces, and various tissues. From 75.5% to 93.3% of the 2,4-D (mainly as parent compound) was excreted within 144 hours (most within the first 24 hours). At least one unidentified metabolite was also found in the urine. Similar results have been reported by Clark et al. (1964) on sheep, and by Lisk et al. (1963) on steer.

Khanna and Fang (1966) noted that the time necessary to eliminate 2,4-D from the body was dose-dependent. Rats eliminated 1 to 20 mg within 24 hours, but 100 mg required 144 hours for a 75% recovery. However, Fang et al. (1973) showed that assimilation efficiency does not appear to be affected by the size of the dose.

Erne (1966) noted that the amine and alkali salts of 2,4-D were readily absorbed in the gastric region, but that the ester was incompletely absorbed. The EPA (1977) noted that all cholorophenoxy acids, salts, and esters were absorbed across the gut wall, the lung, and skin, but were not stored in significant amounts in fat. Excretion usually occurred within hours, or at most days, usually in the urine. Erne (1966a, b) stated that the plasma half-life of 50-100 mg/kg of orally introduced 2,4-D was 3 to 12 hours in rats, pigs, and calves. Pigs eliminated the 2,4-D mainly in the urine, primarily as the parent

compound, but also as unidentified acid-hydrolyzable conjugates. Penetration into the central nervous system and adipose tissue was restricted, but placental transfer was rapid in pigs.

Fang et al. (1973) reported that small amounts of phenoxy herbicides were passed to the young through their mother's milk. The diet-to-milk concentration factor, however, was less than 10 to 3 (Bjerke et al., 1972).

Elo and Ylitale (1977) introduced a subcutaneous dose of 250 mg/kg into rats. Within 4.5 hrs, 67% of it was located in the plasma. A breakdown product, 2,4-dichlorophenol, was identified. H. E. Christensen et al. (1974) gave an oral LD_{50} value of 580 to 1625 mg/kg for mice and rats for this substance.

Graff et al. (1972) injected 250 mg of 2,4-D intraperitoneally and reported that there was a reduction in the synthesis of acid-soluble organic phosphate in muscles. The authors concluded that the pathogenic mechanism of 2,4-D was the uncoupling of oxidative phosphorylation.

Special Studies

Carcinogenicity There has been considerable controversy regarding the potential for 2,4-D to cause cancer. Most of the controversy has involved a study by Hansen et al. (1971) who administered 0, 5, 25, 125, 625, and 1250 ppm 2,4-D to male and female Osborne-Mendel rats for 2 years. He noted no adverse toxicological effects or increased incidence of tumors above control values. As part of the same investigation, no increased incidence of tumors was noted in a 2-year dog study in which beagle dogs were given 0, 10, 50, 100, or 500 ppm 2,4-D in their diets.

There is general agreement that the dog study does not show 2,4-D to be a carcinogen. Melvin Reuber, a toxicologist at the National Cancer Institute, qualifies his support of the study by noting that 2 years is an insufficient duration for a dog study. Dr. Reuber's primary

disagreement, however, is in regard to the rat study. Reuber (1979) believes that the histological examination of rat tissues was inadequate in that only grossly visible neoplasms were sectioned, and therefore microscopic neoplasms may have been missed. He also faults the practice of performing detailed histopathology on six rats of each sex in the high dose group and controls, with reduced tissue consideration at other treatment levels. Reuber claims, furthermore, that he reviewed the complete set of raw data and histologic sections. His conclusion is that 2,4-D is carcinogenic in rats.

Reuber's statements regarding inadequacies in the study's methods are justified. However, it must be noted that the experiment was conducted in 1964 and reported in 1971; the state of the art regarding these tests has changed considerably since that time. Regarding his conclusion about the carcinogenic potential of 2,4-D, it is difficult to critique his judgment in the absence of the raw data. Some of his statements, however, are questionable. For example, he notes a dose-related increased incidence of malignant neoplasms for all sites in male rats. His data, however, indicated little change in incidence: 36% (9/25) for the 25 and 125 ppm groups, 25% (6/24) for the 625 ppm group, and 39% (9/23) for the 1250 ppm group.

The Hansen data for rats were reviewed by the National Cancer Institute, the EPA, and the editorial staff of the Journal of Toxicology and Applied Pharmacology, all of whom agreed with the author's conclusions (although probably without a reexamination of the histologic sections). The greatest value in Reuber's criticism may be in pointing out inadequacies in study technique and the need for further study. Therefore, this report concludes that no conclusions regarding the carcinogenic potential in rats can be made on the basis of this study, i.e. it presents no clear evidence for or against the carcinogenic potential of 2,4-D.

In another study, Innes et al. (1969) found no increase in the incidence of tumors above control values in male and female mice (C57BL/6 × C3H/Anf) or C57BL/6 × AKR) mice given an oral dose of

46.4 mg/kg by gavage on days 7-28 of age, followed by an addition of 2,4-D in the diet for approximately 18 months. Dietary additions were 111 ppm for the isopropyl ester, 149 ppm for the butyl ester, and 130 ppm for the isooctyl ester.

Vettorazzi (1975), as reported in a review by Arthur D. Little, Inc. (1979), found no increased incidence of tumor formation in mice fed 2,4-D orally for their lifespan.

Eriksson et al. (1981), in a case-control study, found indications that occupational exposure to phenoxy acids (2,4,5-T, 2,4-D, MCPA, mecroprop, and dichlorprop) and chlorophenols might constitute roughly a six-fold increase in risk for the development of soft tissue sarcomas. As is often true for such studies, the investigation involved a relatively small sample size. It is difficult to draw conclusions regarding the effect of 2,4-D, separated from the effects of other chemicals to which exposure occurred.

One study showed the proliferation of peroxisomes following administration of 2,4-D. Vainio et al. (1982) showed an increase in the mean frequency of peroxisomes from 17.7/100 μm^2 (controls) to 27.3/100 μm^2 in liver cells of Chinese hamsters given nine daily doses of 100 mg/kg of 2,4-D by gavage. Vainio and his co-workers suggest that 2,4-D may fit into a novel class of compounds that are carcinogens in rodents and whose mechanism of action appears to involve the excessive production of hydrogen peroxide-generating enzymes. Liver cells may thus be exposed to the cytotoxic or DNA-damaging potential of hydrogen peroxide, leading to the subsequent development of liver neoplasia. This peroxisome proliferation response, however, does not occur in humans, monkeys, rabbits, or guinea pigs (Cohen and Grasso, 1981). These results are therefore of questionable relevance to an assessment of risk to humans.

A slight reversible effect was noted in bovine fetal muscle cells exposed in culture to 2 or 20 mg/l of 2,4-D in a study by Basrur et al. (1976). Cultures exhibited an initial drop in total cell counts at 48 hours, but

recovered in 96 hours. The percentage of mitotic cells in cultures treated with the higher dose of 2,4-D was also decreased at 24 hours, but was within normal values at 48 hours.

Teratogenicity/Reproduction There is considerable disagreement over the interpretation of tests that assess the teratogenic/reproductive effects of 2,4-D. Conclusions regarding the results of these tests are generally either that 2,4-D is a "weak teratogen" or that it does not cause "true teratogenic" effects. The review conducted for this report concluded that

1. At very high doses (1000 mg/kg and above), there seem to be teratogenic and/or reproductive effects;
2. At doses below 50 mg/kg, there seems to be little or no teratogenic or reproductive effect;
3. At doses of 50-150 mg/kg, adverse effects occur in some studies and not in others.

There is considerable disagreement among previous reviewers regarding the significance of the effects that occur at 50-150 mg/kg, and also whether they should be considered teratogenic, embryotoxic or fetotoxic. Disregarding the semantic difficulties, the position of this report is that the observed malformations should be considered significant, whether or not they affect the organism's chances of survival. However, the lack of a clear dose response in some studies, the low incidence of abnormalities in most studies, and the absence of any adverse effects in other studies, indicate that 2,4-D can only be considered, at most, a weak teratogen. Furthermore, it should be noted that equivalent doses of 50-150 mg/kg in humans (3.5 to 10.5 g) are highly unlikely even for a worst-case exposure.

The following is a brief discussion of some of the key studies that examine the effects of 2,4-D when administered at doses of 50-150 mg/kg. For further detail (and alternative views) the reader is urged to read two reviews, one by Mullison (1981) and one by the Epidemiological Studies Laboratory of the State of California (1980).

In a study by Collins and Williams (1971), 20 to 100 mg/kg 2,4-D was administered to hamsters on days 6 to 10 of gestation. Occasional abnormalities (usually fused ribs) were noted, along with decreased fetal viability. Neither of these effects was dose related. The number of fetal abnormalities was not statistically significant.

Khera and McKinley (1972) noted a slight increase in fetopathology and incidence of fetal skeletal anomalies in pregnant Wistar rats at levels of 100 or 150 mg/kg/day. Effects included delayed ossification and wavy and lumbar ribs. The 2,4-D was administered in the form of isooctyl and butyl esters and the butoxyethanol and dimethylamine salts. No adverse effects were noted at 25 and 50 mg/kg/day. Similar skeletal abnormalities were observed by Schwetz et al. (1971) at doses of 50 mg/kg and above. The effects were dose-related. No significant adverse effects were noted at doses of 12.5 and 25 mg/kg.

No evidence of embryo or fetal lethality or maternal toxicity was seen in groups of CD rats given daily oral doses of either the propylene glycol butyl ether or isooctyl esters of 2,4-D at molar equivalents of 0, 6.25, 12.5, 25, or 87.5 mg 2,4-D/kg/day on days 6 through 15 of gestation. No gross or soft tissue anomalies were observed. An increased number of fetuses in the two 87.5 mg groups had fourteenth rib buds, but this observation was considered to be within the normal range of variation. Postnatal growth and survival of pups in the 87.5 mg groups were not adversely affected (Unger et al., 1981).

Courtney (1977) noted an increase in the percentage of cleft palates when 0.56 and 1.0 mM/kg (approximately 124 and 221 mg/kg) 2,4-D was administered to CD-1 mice. Cleft palate occurred in 5%-16% of the mice (compared to 0% in the control group) for two esters and the acid. Cleft palate was observed when oil or DSMO was used as a carrier, but not when sucrose was used as a carrier.

Konstantinova et al. (1976) noted no adverse effects on embryos of random-bred female rats given either 2,4-D (0.1, 1, or 50 mg/kg) by gavage during gestation. The 2,4-D was given daily through day 20 of

gestation. Because of discrepancies between the text and tables, the exact results are difficult to determine. However, it appears that 50 mg/kg induced an increased incidence of fetal hemorrhages and that no effect was seen at lower doses.

In a three-generation rat reproduction study (Hansen et al., 1971), Osborne-Mendel rats were given 100, 500, and 1500 ppm of 2,4-D. At the highest dose, the body weights of weanlings were markedly reduced, as was the percentage of pups that survived to weaning. These effects were not observed at 100 and 500 ppm. No effects on fertility or litter size were observed at any dose. No terata were reported; however, there was no specific examination for teratogenic effects.

Carmelli et al. (1981) found no positive association between occupational phenoxy herbicide exposure in males and subsequent spontaneous abortions in their wives. A suggestive association with overall 2,4-D exposure was noted in an isolated subgroup of wives of young forest/commercial workers, but not for the same age group of farmer's wives, indicating that the suggestive association may be attributable to chance alone.

Mutagenicity Although there is conflicting data, the most reliable tests indicate that 2,4-D is not a mutagen. As indicated in Table II-14, no mutagenic response was found in experimental data that measure heritable genetic lesions in whole animal bioassays. Both positive and negative findings are observed in the remaining battery of cellular/ in vitro studies. Positive findings in one or more of these tests, though they may suggest the possibility of heritable genetic lesions, are insufficient to outweigh the findings of the whole animal bioassays.

TABLE II-14

MUTAGENICITY TESTS: 2,4-D

Test System	Result	Comments	Sources
Mouse dominant lethal	-	5 oral doses 75 mg/kg	Epstein et al. (1972)
	-	Single i.p. injection of 125 mg/kg	"
Drosophila sex-linked recessive lethal	-		Gopalan and Njagi (1981)
Mouse bone marrow in vivo	+	100-300 mg/kg orally	Pilinskaya (1974)
Human lymphocytes in vitro	+	50 µg/ml chromosome deletions	Korte and Jalal (1982)
	+	10 µg/ml; sister chromatid exchange	"
	-	0.2 µg/ml	"
	+	0.1-250 µM; chromosomal aberrations	Pilinskaya (1974)
	-		Fahrig (1974)
Chinese hamster V79 cells	+	0.01 mM	Ahmed et al. (1977)
Host-mediated assay Mouse/Salmonella)	-		Zetterberg et al. (1977)
Mouse/Saccharomyces)	-		"
Saccharomyces cerevisiae D4 Mitotic recombination	+	0.1-0.75 mg/ml	Zetterberg et al. (1977)

TABLE II-14 (CONTINUED)

MUTAGENICITY TESTS: 2,4-D

Test System	Result	Comments	Sources
Saccharomyces marcescens	-		Fahrig (1974)
Bacillus subtilis "Rec" assay	-		Shirasu et al. (1976)
Escherichia coli WP2	-		Moriya et al. (1983)
	-		Shirasu et al. (1976)
	-		Probst et al. (1981)
E. coli DNA cell-binding	+	20-200 µM, only in presence of liver extract	Kubinski et al. (1981)
Ames/Salmonella	-		Shirasu et al. (1976)
	-		Moriya et al. (1983)
	-		Zetterberg et al. (1977)
	-	5 µl	Anderson et al. (1972)
rII mutants of T_4 bacteriophage	-	50 µg	Anderson et al. (1972)
Unscheduled DNA synthesis, rat hepatocytes	-	1000 nmoles/ml	Probst et al. (1981)
Pelargonium zonale	+		Pohlheim et al. (1977)

Dioxin Contamination of 2,4-D

Cochrane et al. (1980) analyzed 58 samples of 2,4-D representing 1980 supplies available in Canada. Ester and amine formulations were found to contain dioxins in the form of 2,7-dichlorodioxin,1,3,7-trichloro-dioxin, and 1,3,6,8-/1,3,7,9-tetrachlorodioxin. About 30% of the amine formulations were found to contain one or more of these dioxins in concentrations ranging from 5 to 587 ppb total dioxin. About 95% of the esters contained one or more dioxins in concentrations ranging from 35 to 23,815 ppb. Acid formulations did not contain any dioxin. The level of detection in this study was 1 ppb.

Internal EPA correspondence (R. Harless, Health Effects Research Laboratory, 1981) reports results on laboratory analysis of 2,4-D for dioxin contamination. In this investigation, three samples previously found to contain some dioxin were analyzed and were found to contain 2,7-dichlorodioxin (2,7-DCDD) at concentrations of 73.5 to 184 ppb, along with other dichlorodioxin isomers at lower concentrations. Two out of the three samples were found to contain 1,3,6,8-tetrachlorodioxin at concentrations of 3 and 5.5 ppb. Other tetrachlorodioxin isomers were found at lower concentrations, but no 2,3,7,8-isomer was detected (detection limit not given). Trichlorodioxins were not analyzed, due to lack of an adequate standard.

The dioxin isomer that has been the subject of considerable attention because of its high toxicity is 2,3,7,8-tetrachlorodioxin. As stated above, this has not been found in 2,4-D. Limited information is available on other dioxins. One 2-year mouse study (NCI, 1979) found some suggestive evidence of carcinogenic potential of 2,7-dichlorodioxin in male mice fed 5000 and 10,000 ppm in their diet. A dose-related incidence ($p = 0.008$) of hepatocellular adenomas or carcinomas were found in male mice, although the report notes a historical incidence of this lesion in the strain of mice used in the experiment (B6C3F1). In male mice, significant increases were found at low doses, but not at high doses, in the incidence of combinations of leukemia and lymphomas and combinations of hemangiosarcomas and hemangiomas. No evidence of tumor induction was noted in female mice.

As part of the same study, Osborne-Mendel rats (35 of each sex) were fed 5000 and 10,000 ppm 2,7-DCDD over a 2-year period. No induction of tumors was noted in males or females.

Khera and Ruddick (1973) of the Canadian Department of National Health and Welfare, performed a teratology test on 2,7-dichlorodioxin. Pregnant Wistar rats were treated orally on days 6 through 15 of gestation with 250-2000 µg/kg/day of 2,7-TCDD in an anisole-corn oil carrier. At higher doses (100-2000 µg/kg) the occurrence of myocardial lesions was noted. (At 1000 and 2000 µg/kg, the number of fetuses with lesions of the myocardium was 2 and 7, respectively. No such lesions occurred at lower doses or in the control group.) No effect was observed at any dose in litter size, resorptions, fetal weight, or skeletal formation, nor in pup weight, growth or survival. It should be noted that the authors did not mention the incidence of myocardial lesions in their abstract but stated, rather, that the substance "produced no significant effects." It is possible that the myocardial lesions may be a toxic response, since growth of cardiac tissue was suppressed and the lesions occurred at very high doses for dioxins. Dioxin is introduced into the environment at concentrations that are several orders of magnitude lower than the herbicide active ingredient. The lack of response at 500 µg/kg may therefore represent an adequate margin of safety for teratogenesis.

Summary An evaluation of important toxicity data is provided at the end of this chapter.

3. MOBILITY AND PERSISTENCE

Fate in Soil

A considerable amount of study has been given to the fate of 2,4-D in soil. This section is divided into separate discussions of leaching/adsorption, runoff, persistence/degradation, and volatilization.

Leaching/Adsorption Data on the mobility of 2,4-D in soil is somewhat conflicting. The overall variability in results may be due to differences

in soil characteristics, since the percentage of organic matter strongly influences 2,4-D mobility, as does clay content to a lesser extent. Also, some studies show that 2,4-D mobility varies with the form of the herbicide, i.e., whether it is in the form of an acid, a salt, or an ester.

The available field studies regarding the leaching of 2,4-D show both high and low mobility. High mobility was shown by a study in which both the 2,4-D amine and the ester were applied to silty loam soil (application rates not given), followed 1 day later by "simulated rainfall." After 2 days, 2,4-D had leached to a depth of 24 cm; after 5 days, it had moved to a depth of 40 cm. Thirty days after application it continued to move downward in the soil (depth not given) (Wilson and Cheng, 1976).

Low mobility was shown by Burcar et al. (1966), who found that the isooctyl ester of 2,4-D (after being hydrolyzed to the acid in the soil) remained for the most part in the upper 5 cm of soil, with no lateral migration observed. Similar results were found by Smith (1975), who found only negligible amounts of 2,4-D below 5 cm at the end of one growing season.

Moderate mobility is indicated by the results of a study by Bornett et al. (1967) who found that 2,4-D esters moved to a depth of 15 cm (only trace amounts were found below this level), although most of the 2,4-D remained in the top 8 cm. Penetration into the 8 to 15 cm layer was greater for the amine salts than for the esters.

In a study by the United States Air Force Academy (Young et al., 1974) extremely high amounts of 2,4-D and 2,4,5-T butyl ester mixtures were applied to Utah soils at a depth of 10 to 15 cm. After 282 days, residues were found throughout the 91 cm core sample. However, 90% of the residues had moved only 15 to 20 cm downward (i.e., they were found in the top 30 cm of the soil profile).

Laboratory studies indicate a greater mobility for 2,4-D than do field studies. The National Research Council of Canada (NRCC) stated in its

review of 2,4-D that recent studies (Eshel and Warren, 1967; Helling, 1971a, b, c; Helling and Turner, 1968; Benson and Covey, 1974; Grover, 1977a) show acid herbicides, including 2,4-D, to be "quite mobile" in the soil. No details regarding these studies were provided. According to Helling's classification (which ranks pesticides according to their mobility), 2,4-D was given a rank of 4, with 5 being the most mobile class. Surfactants were found to increase the mobility of 2,4-D (Helling, 1971b). De Rose (1946) found that 2,4-D moved readily through greenhouse soil. The NRCC review (1978) cautioned that some of these laboratory column studies do not allow time for equilibration of the adsorption/desorption process. However, Norris (1970) found that adsorption and desorption processes proceeded at roughly equal rates, attaining equilibrium rapidly within 180 minutes.

In laboratory studies low mobility was found by Crafts (1949) and by Nutman et al. (1945), although no details are available on these studies.

Some of the variability in results may be due to formulation. Although NRCC (1978) states that all salts and esters of 2,4-D will be hydrolyzed to the acid in moist soil, the potential for leaching does seems to vary with the form of the herbicide. When Wiese and Davis (1964) applied 2,4-D to columns of silty clay loam soil, it was found that the alkanolamine salt, which is 100% soluble in water, moved 15 inches, while the butoxyethanol ester (with a solubility of 16 ppm) leached 3 inches. The lower mobility of the ester forms (as compared to the salt or acid forms) was confirmed by others (Smith and Ennis, 1953; Aldrich and Willard, 1952; Barnett et al., 1967).

The type of soil, particularly the amount of organic matter, may also account for the variability in results in studies of the mobility of 2,4-D. Ogle and Warren (1954) showed that 2,4-D had low mobility in muck but leached readily in mineral soils. Hernandez and Warren (1950) showed that the sodium salt of 2,4-D leached 7.5 cm in a peat soil and 13 cm in a soil with low organic content after 10 cm of water had been applied. Several investigators have attempted to correlate soil

properties with adsorption of 2,4-D; correlation has been found only with the percentage of organic matter in soil (Hamaker et al., 1966; Grover, 1973 and 1977; Grover and Smith, 1974; O'Conner and Anderson, 1974; Liu and Cibes-Viade, 1973). Acid herbicides, including 2,4-D, have been found to adsorb readily to soil organic fractions (Harris and Warren, 1964; Grover and Smith, 1974). Two studies have shown a high degree of negative correlation between phytotoxicity and organic matter content of soil (Meadows and Smith, 1949; Upchurch and Mason, 1962).

The strength of the adsorption of 2,4-D to organic matter is unclear. On the one hand, two studies have shown that 2,4-D is held tightly to organic matter; attempts to desorb the herbicide with water have yielded limited recoveries. (Harris and Warren, 1964; Grover, 1977). On the other hand, Norris (1970) found that both adsorption and desorption of 2,4-D were rapid on forest soil material, suggesting a low energy of adsorption. Support for this idea comes from equilibrium and kinetic studies done by Hague and Sexton (1968) and by Khan (1973). These studies found that the interaction of 2,4-D and humic acid was a relatively weak one, with physical rather than chemical forces holding the 2,4-D in the interior spaces of the humic acid.

Most investigations show that 2,4-D adsorbs only weakly and in small amounts to clay particles (Harris and Warren, 1964; Scott and Lutz, 1971; Grover, 1977; Coffey and Warren, 1969). This is to be expected, given the anionic nature of most of the dissociated forms of the 2,4-D, and the negative charge of the surfaces of clay particles. Although some adsorption can occur at pH levels of 3 or below, 2,4-D adsorption onto clay at normal soil pH is nil or even negative (Frissel, 1961; Frissel and Bolt, 1962).

Runoff The few studies available suggest the potential for 2,4-D movement in runoff may be significant. When 11.2 kg/ha and 1.1 kg/ha of the amine and ester were applied to 16%-17% slopes, the amounts of the 2,4-D ester collected in the runoff water were 3.4 ppm

and 2.0 ppm for the two rates of application, respectively. The amounts of the 2,4-D amine collected in the runoff water were 4.5 pm and 2.0 ppm (Wilson and Cheng, 1976). In a study using the isooctyl ester of 2,4-D, Barnett et al. (1967) found that the amount lost exceeded 10% of the amount applied, and that the highest concentrations occurred in the first 15 minutes of runoff during a rainstorm. TRW (1981) suggests that the less water-soluble forms may have more potential for runoff because of their tendency to be held at the surface of the soil. The TRW report cites a study by Tarrant and Norris (1967) in which artificial rain was applied to a test plot of sandy loam soil. The results showed that 3% of the relatively water-soluble amine was lost in runoff, while 27% of the less-soluble ester was lost. (This negative correlation between solubility and runoff is contradicted by the results of Wilson and Cheng (1976), mentioned above.)

A study by Douglas et al. (1969) showed that untreated strips of vegetation on either side of water channels could minimize contamination by 2,4-D.

Persistance/Degradation There is general agreement that 2,4-D can be considered a non-persistent herbicide (Newman and Thomas, 1950; Norris, 1966, 1967, 1970; Helling, 1971; NRCC, 1978). It is extensively degraded (85%-90%) in 15 days in many soils (Freed and Montgomery, 1963; Hernandez and Warren, 1950; Loos, 1969; Norris, 1966, 1970, 1971).

The half-life of 2,4-D in forest-floor material was found to be 10 days in a field study (Norris and Moore, 1971) and 4 days in a laboratory study (Altom and Stritzke, 1973). Longer half-lives (14-41 days) were found in a study using Saskatchewan soils (Foster and McKercher, 1973). Table II-15 documents the various studies and the percentages of the amount applied that remained after a specific length of time.

TABLE II-15

PERSISTENCE OF 2,4-D IN SOIL

Soil Type	Type of Study	Formulation	Application Rate	Percentage Remaining	Length of time (days)	Source
Forest soil	Lab	NS	NS	50	4-5	Altom and Stritzke (1973)
Red alder forest floor material	NS*	NS	2.24 kg/acre	6	35	Norris (1970b)
Silty loam soil	NS	Free phenoxy acid	7.8, 15.7 kg/ha	1	70	Stewart and Gaul (1977)
		" " "	31.4 kg/ha	2	70	
		Isooctyl ester	7.8, 15.7, 31.4 kg/ha	ND	14	
		Amine	(same)	<5	70	
NS	Field cond.	Ester	NS	ND**	14	Burcar et al. (1966)
Soils at or above wilting point	NS	Isobutyl ester		ND	1	Smith (1972c, 1976a)***
		Isopropyl ester	NS	ND	1	
		Isooctyl ester	NS	ND	3	
Acid soil, forest clearing	Field	NS	"Heavy"	10	105	Flieg and Pfaff (1951)

*NS = not specified.
**ND = None detected.
***Rapid hydrolysis of esters.

TABLE II-15 (CONTINUED)

PERSISTENCE OF 2,4-D IN SOIL

Soil Type	Type of Study	Formulation	Application Rate	Percentage Remaining	Length of time (days)	Source
Saskatchewan soils	Lab (26°C)	NS	NS	50	14-41	Foster and McKercher (1
NS	Greenhouse soybean assays	NS	25 mg/kg soil	ND	56	Burger et al. (1962)
Unspecified field soil		NS	5.5 kg/ha	ND	49	DeRose and Newan (1948)
			22 kg/ha	ND	93	
NS	NS	NS	NS	0-25	∿30	Kearney (1966)
Red alder forest floor material	NS	NS	NS	55	10	Norris and Moore (1971)
				16	20	
				5	35	

*NS = Not specified.
**ND = None detected.

Degradation of 2,4-D is predominantly microbial, rather than chemical, and numerous bacteria have been isolated that are capable of degrading 2,4-D. Some of the these are listed in Table II-16, along with the identified reaction products. The degradation of 2,4-D has been well studied and can be roughly summarized in the following steps:

1. Esters and amide formulations are first hydrolized by an enzymatic or a soil-catalyzed reaction (Norton, 1975; Smith, 1972).
2. The acetic acid side-chain is removed to yield the corresponding phenol.
3. A ring cleavage results in an aliphatic acid (TRW, 1981; NRCC, 1978; USDA, 1973).

Aliphatic acids (e.g., succinic acids) are common soil constitutents which microorganisms can use as carbon sources, thereby releasing the original material as CO_2.

Volatilization Little information was found on the potential for loss of 2,4-D by volatilization from the soil surface, even though some forms of the herbicide are highly volatile. Based on limited data, TRW (1981) suggested that volatility may affect the rate of initial loss but that it would have an insignificant effect on long-term persistence.

Persistence in Water Several monitoring studies have investigated residues of 2,4-D primarily from agricultural areas. In a survey of 20 rivers in the western United States, the U.S. Geological Survey found 40 of 331 water samples to contain 2,4-D in concentrations of 0.03 to 0.35 mg/l (Manigold and Schulze, 1969). Considerably lower concentrations were found in water from eight agricultural watersheds in Ontario: 2,4-D was present in 39% of these samples, with a mean concentration of 0.2 µg/l, with a range of <0.1-16 µg/l (Frank et al., 1978). These same investigators studied 11 agricultural mini-watersheds and found that 38 of 404 samples (9.4 %) contained 2,4-D. Although in most samples (33 of the 38) the residual level was less than 1 µg/l, one contained 16 µg/l and another 320 µg/l. The investigators explained the very high concentration as the result of spraying a nearby right-of-way at the time of sampling. Other information on residues is presented in Table II-17.

TABLE II-16

DEGRADATION OF 2,4-D BY MICROORGANISMS

Species	Main Reaction Product	Other Reaction Products	References
Pseudomonas sp.	β-chloromuconic acid		Fitzgerald (1966)
"	α-chloromuconic acid		"
"	6-hydroxy-2,4-D		Loos et al. (1967)
Achromobacter sp., Nocardia spp.	2,4-dichlorophenol	Chlorohydroquinone, monochlorophenol, unchlorinated phenol, 3 unidentified compounds	Audus (1950); Bell (1957, 1960); Faulkner and Woodcock (1964); Norris (1966); Steenson and Walker (1957, 1958) Taylor and Wain (1962)
Aspergillus niger	2,4 dichloro-5-hydroxy-phenoxy acetic acid	2,4-dichloro-4-hydroxy-phenoxyacetic acid unidentified form	Faulkner and Woodcock (1965); Fawcett et al. (1954)
Athrobacter sp.	2,3 dichloranisole	2,4-dichlorophenol, chloride	Loos, et al. (1967)
Corynebacterium sp.	Hydrolyzed 2,4-D	Respective alcohols, chlorides	Aly and Faust (1964)

TABLE II-17

RESIDUES OF 2,4-D IN WATER

Sampling Area	No. of Samples	No. of Samples Containing 2,4-D	Range of Concentration	Comments	Source
Western U.S. Rivers	331	40	0.03-0.35 mg/l		Manigold and Schultz (1959)
8 Agric. watersheds, Ontario	-	39%	<0.1-1.6 µg/l		Frank et al. (1978)
11 Agric. miniwatersheds, Ontario	404	38	<1-320 µg/l	Highest conc. due to spraying just prior to sampling	Frank et al. (1978)
Saskatchewan River	-	-	0.4-22 µg/l	Samples taken during spraying season	Choi et al. (1976)
Guntersville Reservoir of Tennessee River	-	2	2-11 µg/l	Sampling done 6 months after spraying at rates 22.4-44.8 kg/ha	Wojtalik et al. (1973)

TABLE II-17 (CONTINUED)

RESIDUES OF 2,4-D IN WATER

Sampling Area	No. of Samples	No. of Samples containing 2,4-D	Range of concentration	Comments	Source
Mud in water body	-	-	0.24-58.8 mg/kg	Residues in mud prior to spraying: 0.14 ppm	Smith and Isom (1967)[3]
Farm ponds in Ontario	-	48%	6-11 µg/l	See text	Frank (1970-1974)
Farm wells in Ontario	-	49%	3-12 mg/l		"
Streams in forested areas of Oregon	-	-	0.001-0.84 mg/l	Within 10 hours of spraying at rates 2.24-3.36 kg/ha	Norris (1967)
	-	-	0.076-0.176 mg/l	Downstream sampling pt.(1/4 mile from sprayed area)- 1 hr after spaying	
Western Oregon streams	-	-	<0.1 ppm	6 yrs of monitoring	Norris (1971)
11 Western U.S. streams	132	0	ND	None found during first year	Brown and Nishioka (1967)

TABLE II-17 (CONTINUED)

RESIDUES OF 2,4-D IN WATER

Sampling Area	No. of Samples	No of Samples containing 2,4-D	Range of concentration	Comments	Source
Perimeter canal in Loxahatchee Nat. Wildlife Refuge (Florida)	-	-	ND	No accumulation in water over 4 months after spraying	Schultz and Whitney (1974)
Nine ponds in Florida, Missouri, Georgia	-	-	0.08-0.69 mg/l in water	Sampling 1-14 days after treatment	Schultz and Harmon (1974a)
	-	-	0.005-0.17 mg/kg in mud		
Irrigation water	-	-	25-61 ppb	Applic rate: 1.9-3 lb/acre	Frank et al. (1970)a
	-	-	Negligible	20-25 miles downstream from applic. pt.	
North Carolina watershed	-	-	1800 ppb, 40 ppb	Applic. rate: 4 lb/acre	Sheets & Lutz (1972)

Once present in the water, 2,4-D may be detectable for 3 weeks to 4 months (Schultz and Harmon, 1974; Wojtalik et al., 1971; Frank and Comes, 1967). Microbial degradation does not appear to be significant in most waters (TRW, 1981; NRCC, 1978), unless the system is warm, aerobic, 2,4-D-rich, and nutrient-rich (Halter, 1980). These conditions are not typical of most natural surface waters in Massachusetts. No breakdown of 2,4-D occurred in lake waters aerobically incubated in the laboratory for 120 days (Aly and Faust, 1964). Watson (1977) and Schwartz (1967). Both documented that 2,4-D is stable in water for up to 6 months, depending on the microorganisms present, nutrient levels, and amount of suspended sediments.

Chemical hydrolysis rates vary considerably with the form of 2,4-D. At pH 6, the hydrolysis half-lives range from 26 to 220 days for several of the esters at 25°C (Zepp, 1975). For some forms of 2,4-D, volatilization may be more important than hydrolysis at pH 6. Half-lives for vaporization at pH 6. Half-lives for vaporization of the butyl, octyl, and methyl esters are 1.1, 11.5, and 21.7 days, respectively.

Some photodecomposition of 2,4-D may take place at the surface of water, although it is minimized in well-mixed deep ecosystems (Zepp et al., 1975; Leighton, 1961). Adsorption onto the suspended particles does not appear to be a significant removal mechanism, according to a review by Halter (1980).

Indicators of Potential Ground Water Contamination

Table II-18 provides information on parameters associated with the mobility of 2,4-D. These parameters, and their associated thresholds, have been suggested by EPA for use in assessing the potential for pesticide contamination of ground water. A discussion of these parameters and thresholds, and the methods for arriving at designated values for individual herbicides, is presented in the main body of the report as part of the discussion of the fate of herbicides in the environment.

TABLE II-18

INDICATORS OF POTENTIAL GROUND WATER CONTAMINATION:
2,4-D

Indicator	Value for 2,4-D	Threshold
Solubility	Insoluble to infinitely soluble, depending on form	>30 ppm
K_{oc}	66-307	<300-500
Speciation at pH 5	Anionic	Anionic (negatively charged)
Hydrolysis half-life	Rapid to slow, depending on form	>6 months
Photolysis half-life	16-29 days*	>3 days
Vapor pressure	6.0×10^{-6} mm Hg at 25°C	$<10^{-2}$mm Hg

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* For dechlorination of the ester.

4. TOXICITY TO NON-TARGET ORGANISMS

Birds

Except for one study on reproduction, a number of tests show 2,4-D to be non-toxic to test birds. Heath et al. (1972) and Hill et al. (1975) reported LC_{50} values for mallard ducks, bobwhite quail, Japanese quail, and pheasants to be >5000 ppm for 2,4-D acetamide, butoxyethanol ester, and dimethylamine salt. Tucker and Crabtree (1970) reported LD_{50} values that ranged from >>100 mg/kg to approximately 2000 mg/kg for mallard ducks exposed to 2,4-D technical acid, technical sodium salt, and 4 lb acid equivalent/gallon of amine. These data are summarized in Table II-19.

Studies by Somers et al. (1972, 1974a, b, c) reported that spraying eggs with 2,4-D and 2,4,5-T, alone and together, resulted in no adverse effects on hatching chicks or embryos. An additional study by Kopischke (1972) found no significant effect of 2,4-D on the hatchability of pheasant eggs.

Two studies by Hilbig et al. (1976a, b) found that spraying eggs of quail, pheasants, and chickens with 2,4-D, in concentrations up to 10 times the recommended doses, produced no effect on the hatching rate, body weight, sexual differentiation, reproductive performance (as adults), or number of malformed chicks. On the other hand, in a study by Lutz-Ostertag and Lutz (1970) which investigated the effects of spraying 2,4-D amine at a concentration of 1.1 kg a.i./ha on fertile eggs (in an artificial nest), they found 77% of the ring-necked pheasant, 43% of the red partridge, and 77% of the grey partridge embryos were dead on the nineteenth day of incubation. Surviving embryos were malformed or partially or completely paralyzed.

Fish

Although toxicity varies with formulation and environmental conditions, DeVaney (1968) concludes that many of the formulations (especially the esters) are toxic to fish. Halter (1980) showed that the acute toxicity of 2,4-D to fish varied considerably, depending on the species of fish, the water quality, and the 2,4-D formulation. A study by Woodward and

TABLE II-19

TOXICITY OF 2,4-D TO BIRDS

Species	Formulation	Test	Result (ppm)	Source
Bobwhite quail	A[1]	LC_{50}	>5000	Heath et al. (1972)
	BEE[2]	"	"	Heath et al. (1972) and Hill et al. (1975)
	DMA[3]	"	"	Heath et al. (1972)
Japanese quail	A	"	"	Heath et al. (1972)
	BEE	"	"	Heath et al. (1972) and Hill et al. (1975)
	DMA	"	"	Heath et al. (1972)
Pheasant	A	"	"	Heath et al. (1972)
	BEE	"	"	Heath et al. (1972) and Hill et al. (1975)
	DMA	"	"	Heath et al. (1972)
	TA	LD_{50}	472	Tucker and Crabtree (1970)
Mallard duck	A	LC_{50}	>5000	Heath et al. (1972)
	BEE	"	"	Heath et al. (1972) and Hill et al. (1975)
	DMA	"	"	Heath et al. (1972)
	TA[4]	LD_{50}	>>100	Tucker and Crabtree (1970)
	TSS[5]	"	>>2025	"
	4A[6]	"	∿2000	"
Pheasants	TA	"	472	"
Pigeons	TA	"	668	"

[1] 2,4-D acetamide [2] Butyloxyethanol ester [3] Dimethylamine salt
[4] Technical acid [5] Technical sodium salt [6] 4 lb acid equivalent/gallon amine

Mayer (1978) showed the effect of temperature on the toxicity of 2,4-D to fish (Table II-20). The data suggest that toxicity increases as the temperature decreases. The authors concluded that neither water hardness nor pH significantly influences toxicity. A study by Schultz (1973), however, suggested that channel catfish and bluegill accumulated more 2,4-D at pH 6 than at pH 9.

It has been observed by several studies that the butyl ester formulations of 2,4-D were many times more toxic than the corresponding acids. Cameron and Anderson (1977) noted that in the field the esters were quickly hydrolyzed to the acid or salt. Cope (1965) noted delays in the spawning of bluegill sunfish for periods of up to 2 weeks after treatment with propylene glycol butyl ether ester (PGBEE) at 5 and 10 ppm. No effects on reproduction or survival of fry were noted.

A study by Schultz (1973) reported that ^{14}C-labeled dimethylamine salt of 2,4-D, at concentrations of 0.5, 1.0, and 2.0 mg/l, produced no mortality or adverse biological effects. It was found that 90% of the residues in the muscles of bluegill exposed to 2.0 mg/l were composed of metabolites of 2,4-D. In studies by Sikka et al. (1977), and by Stalling and Huckins (1978), it was suspected that decomposition of 2,4-D had occurred in the water due to microbial action.

Lower Aquatic Organisms

Two studies investigated the effect of 2,4-D on amphibians. Sanders (1970a) determined the LC_{50} values for 24-hr and 96-hr tests on Pseudoacris triseriata to be 100 mg/l. Cooke (1972) found that 50 mg/l of 2,4-D produced no visible changes or behavioral abnormalities in tadpoles of Rana temporaria.

Elder et al. (1970) found that 2,4-D exhibited low toxicities to all fresh water and marine algal species tested, at concentrations of maximum solubility in water. Hawxby et al. (1977) found no adverse effects on cyanobacteria and algae tested at 0.10 to 10.0 µM (Anabaena variabilis,

TABLE II-20

TOXICITY OF 2,4-D TO FISH

Species	Formulation	Temp. (°C)	Time	Result LC 50 (ppm)	95% Confidence Limit	Source
Cutthroat trout[1]	BE[2]	5	96-hr	.490	(.397-.606)	Woodward and Mayer (pub. date not given)
		10	"	.540	(.460-.640)	"
		15	"	.770	(.657-.902)	"
	PGBEE[3]	5	"	.490	(.400-.604)	"
		10	"	1.030	(.920-1.200)	"
		15	"	.780	(.663-.918)	"
Lake trout[1]	BE[2]	5	"	.600	(.6541-.665)	"
		10	"	.640	(.569-.720)	"
		15	"	.820	(.715-.940)	"
	PGBEE[3]	5	"	.700	(.631-.777)	"
		10	"	.630	(.542-.733)	"
		15	"	1.000	(.824-1.210)	"
Bluegill	PGBEE[3]	not given	24-hr	2.1	-	Hughes and Davis (1963)
	BEE[5]	"	"	2.1	-	"
	BE[2]	"	"	1.3	-	"
	IE[6]	"	"	1.3	-	"

TABLE II-20

TOXICITY OF 2,4-D TO FISH

Species	Formulation	Temp. (°C)	Time	Result: LC_{50} (ppm)	95% Confidence Limit	Source
Rainbow trout	PGBEE[3]	"	"	1.2	-	U.S.D.I. (1964)
Rasbora heteromorpha	BEE[5]	"	48-hr	1.0	-	Alabasto (1969)
Fathead minnow	DMA[7]	"	96-hr	10	-	Lawrence (1966)
	Acetamide	"	"	5	-	"

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[1] pH = 7.2; water was softened with 40 mg/l of $CaCO_3$.

[2] 2,4-D butyl ester.

[3] 2,4-D propylene glycol butyl ether ester.

Lyngbya sp., Chlorococcum sp., and Chlorella pyrenoidosa). They also concluded that 2,4-D was toxic to lower aquatic organisms (TL_{50} = 0.1 to 2.6 ppm) except crayfish.

Many studies have been done to assess the effects of 2,4-D on lower aquatic organisms (Table II-21). The data suggest that toxicity varies with the different formulations of 2,4-D. Rawles (1965) noted that when 2,4-D acetamide was applied at 20 lb/acre to control Eurasian milfoil, it was toxic to blue crabs and eastern oysters. Butyl or isooctyl esters were not toxic to these test animals. The isooctyl toxicities of the different formulations of 2,4-D were also investigated by Sanders (1969, 1970b) and Zimakowska (1973). They concluded that some of the ester formulations were the most toxic.

Indirect Effects on the Aquatic Ecosystem

After an extensive review of literature, the NRCC (1978) concluded that spraying of phenoxy herbicides, including 2,4-D, to control nearby terrestrial plants may cause direct lethal or sublethal effects in fish or aquatic invertebrates. If the concentration is sublethal but high enough to kill aquatic macrophytes, a complex series of secondary changes may occur throughout the ecosystem, resulting in reduced oxygen and pH levels, increased CO_2 levels, and changes in the species composition of invertebrates and phytoplankton. In response to these changes, a number of food webs that they were part of would necessarily be affected.

Mammalian Wildlife

The effects of 2,4-D on mammalian wildlife have been the subject of a number of studies. Shifts in the population size of pocket gophers after spraying with 2,4-D have been reviewed by Tietjen (1973). Johnson and Hansen (1969) studied the effects of range treatment with 2,4-D and the effect on mice, chipmunk, and vole populations. Wilber (1963) followed the effects that spraying 2,4-D esters had on grazing by elk for several years. Deer forage was followed for 6 years after treatment with 2,4-D ethyl ester in a study by Krefting and Hansen (1969).

TABLE II-21

TOXICITY OF 2,4-D TO LOWER AQUATIC ORGANISMS

Organism	Form	Duration	Dose (ppm)	Effect	Source
Oyster	BE[1]	96-hr	3.75	50% decrease in shell growth	Butler (1965a)
	DMA[2]	"	2	No effect on shell growth	"
	EHE[3]	"	5	38% decrease in shell growth	"
	PGBEE[4]	"	1	39% decrease in shell growth	"
Shrimp	BE	48-hr	1	No effect	"
	DMA	"	2	10% mortality	"
	EHE	"	2	10% mortality or paralysis	"
	PGBEE	96-hr	1	39% decrease in shell growth	"
Fish	BE	48-hr	5	TL_m	"
	DMA	"	15	No effect	"
	EHE	"	10	No effect	"
	PGBEE	"	4.5	TL_m	"
Phytoplankton	BE		1	16% decrease in CO_2 fixation	"
	DMA	4-hr	1	No effect of CO_2 fixation	"
	EHE	"	1	49% decrease in CO_2 fixation	"
	PGBEE	"	1	44% decrease in CO_2 fixation	"
Scud	PGBEE	48-hr	2.6	TL_{50}	Sanders (1970)
	BEE[5]	"	5.9	TL_{50}	"

TABLE II-21 (CONTINUED)

TOXICITY OF 2,4-D TO LOWER AQUATIC ORGANISMS

Organism	Form	Duration	Dose (ppm)	Effect	Source
Sowbug	PGBEE	"	2.2	TL_{50}	"
	BEE	"	3.2	TL_{50}	"
Crayfish	PGBEE	"	100	TL_{50}	"
	BEE	"	100	TL_{50}	"
Daphnia	PGBEE	"	0.1	TL_{50}	"
	BEE	"	5.6	TL_{50}	"
Stonefly	BEE	96-hr	1.6	LC_{50}	Hinckley (1972)
	BE	"	8.5	LC_{50}	"
	BEE	24-hr	8.5	LC_{50}	Sanders and Cope (1968)
	BEE	48-hr	1.8	LC_{50}	"
	BEE	96-hr	1.6	LC_{50}	"
	Technical 2,4-D	24-hr	56	LC_{50}	"
	"	48-hr	44	LC_{50}	"
	"	96-hr	14	LC_{50}	"

[1] 2,4-D butyl ester
[2] 2,4-D dimethylamine
[3] 2,4-D ethyl hexyl esters
[4] 2,4-D proylene glycol ether ester
[5] 2,4-D butoxyethanol ester

In a review of these studies, NRCC (1978) concluded that the applications did not reach toxic levels for any of these species, and that the effect on vegetation resulted in an increase in food availability for voles, elk, and deer, and a decrease in the food source and cover for gophers and chipmunks. The populations of voles, elk, and deer increased in size, but populations of gophers and chipmunk decreased in size. The population of mice remained relatively stable, probably due to their variable diet.

Palmer and Redeleff (1969) report that the acid of 2,4-D fed to mule deer for 30 days at 86 and 240 mg/kg/day produced only minor symptoms and no weight loss. Tucker and Crabtree (1970) report an LD_{50} of 400-800 mg/kg of the 2,4-D acid for mule deer.

Livestock

Livestock do not appear to be sensitive to 2,4-D (Table II-22). In a review of the literature, NRCC (1978) also concluded that there was little direct hazard of toxicity to livestock, but went on to suggest that deaths of domestic animals may be linked to changes in plant chemistry due to treatment with the herbicides. Frank and Grigsby (1957) and Buck et al. (1961) have reported variable effects of 2,4-D on the nitrate concentration of various plants. Nitrate poisoning of livestock has been reported by Fertig (1953). Nitrate accumulation appeared to be species-specific: some species increased while others decreased their nitrate concentration. Phenoxy herbicides (including 2,4-D) may also be associated with increases in the alkaloid and hydrogen cyanide concentration of some plants (Swanson and Shaw, 1954; Lynn and Barrons, 1952; and Willard, 1950). In their review, NRCC (1978) concluded that the significance of any chemical changes and their potential risk to domestic animals was difficult to assess because the data were sometimes contradictory or incomplete, and often inconclusive.

Insect Predators and Parasites

The only study presently available is a study by Adams (1960) in which coccinellid larvae were treated with 2,4-D amine. A four-fold increase

TABLE II-22

TOXICITY OF 2,4-D TO LIVESTOCK

Formulation	Animal	Dose	Duration	Effect	Source
Triethanolamine	Swine	50 mg/kg/day	3 doses	None	Bjorklund and Erne (1966)
	"	"	8-10 doses	Minor transient	"
	"	500 ppm in feed	1 month	Some locomotor disturbances; reduced growth rate; no gross pathology	"
	Chicken	1000 ppm in water	Daily from hatching to 2 months of egg production	Egg size normal; 30% reduction in production	"
Butyl ester	Swine	50 mg/kg/day	5 doses	None	"
Alkanolamine	Sheep	100 mg/kg/day	481 days	No effect	Palmer and Radeleff (1969)
	Cattle	50 mg/kg/day	112 days	"	"
	Chicken	100 mg/kg/day	10 days	No effect on weight gain	Pimentel (1971)
	Chick	380-756 mg/kg		LD_{50}	Rowe and Hymas (1954)
Propylene glycol	Sheep	100 mg/kg/day	481 days	No effect	Palmer and Radeleff (1969)
Butylether ester	Cattle	100 mg/kg/day	10 days	No effect	"
	Chicken	50 mg/kg/day	10 days	No effect on weight	Pimentel (1971)

TABLE II-22 (CONTINUED)

TOXICITY OF 2,4-D TO LIVESTOCK

Formulation	Animal	Dose	Duration	Effect	Source
Ethyl hexyl ester	Cattle	250 mg/kg/day	14 days	Ill in 3 days; survival and recovery from 9 doses; 14 doses lethal	Mitchell et al. (1946)
	Sheep	250 mg/kg/day	17 days	As above; 17 doses lethal	"
	Sheep	100 mg/kg/day	10 days	None to minor effects	"
	Cattle	100 mg/kg/day	10 days	None to minor effects	"
Isopropyl ester	Chick	1420 mg/kg		LD_{50}	Rowe and Hymas (1954)

in mortality was reported, along with an increase in pupation time. Little mortality was seen among the adults. Most adults recovered after a period of inactivity.

Bees

Atkins et al. (1975) concluded that field applications of 1 kg/ha would produce a dosage of 1.12 µg/bee and would be relatively non-toxic to bees. Johansen (1959) reported that 2,4-D was non-toxic to bees except when formulated as a alkanolamine salt or isopropyl ether. Moffett and Morton (1975), Morton et al. (1972), and Moffet et al. (1972), reported 2,4-D to be relatively non-toxic to bees (LD_{50} = 0.015 mg/l) except when diesel oil was used as a carrier (resulting in high mortality). Moffett and Morton (1975) also reported that drownings occurred when surfactants were added to drinking water.

Soil Organisms

Bauer (1961) noted that 2,4-D was very susceptible to breakdown by soil microorganisms and that it showed no signs of impact on the soil microbes at normal application rates. In a report by NRCC (1978), it was concluded that at field concentration, phenoxy herbicides have no dramatic effect on soil microbes. Magee and Colmer (1956) found that the rate of oxygen absorption by three species of Azotobacter (a nitrogen-fixing bacterium) was unaffected when they were exposed to 2000-5000 mg/kg 2,4-D amine. Teater et al. (1958) reported that significant accumulations of nitrate occurred when 2,4-D amine was added to incubated soil at 8.8 and 35 kg/ha, and that stimulation of carbon dioxide evolution was significantly affected only at 35 kg/ha.

Gaur and Misra (1972) cultured seven species of the nitrogen-fixing bacterium that grows symbiotically in legume root nodules, Rhizobium, in broth with 50-2000 mg/l 2,4-D. Growth of one of the species was stimulated by 250 mg/l and above; two of the species were slightly suppressed by 250 mg/l and above; and two other species were significantly suppressed at 250 mg/l and above. The effect on the other two species in the study was not discussed.

Balasubramanian and Rangaswami (1973) concluded that the concentration of bacteria, fungi, and actinomycetes populations increased in number in the rhizospheres of sorghum sprayed with 2,4-D. Root exudations of sugars and amino acids also increased.

Dow Chemical U.S.A. (1972) investigated the effect that 2,4-D had on earthworms. They found that no mortality was produced when worms were immersed in 0.1, 1.0, 10.0, and 100 ppm of 2,4-D for 2 hours. 1000 ppm produced 100% mortality. They also found that there was no effect on wireworms, springtails, mites, or other micro-arthropods at typical field concentrations.

Bioaccumulation

Lowe (1964, cited by Rawls, 1971) found that the acid or ester of 2,4-D disappeared rapidly from the tissues of fish and oysters when exposure was discontinued. Studies by Erne (1966) concluded that 2,4-D amine and alkali salts were not retained in tissues of pigs, chickens, or calves, even following repeated administration. On the other hand, Rodgers and Stalling (1977) and Shultz and Whitney (1974) showed that fish accumulate residues that were unidentified metabolites of 2,4-D. Stalling and Huckins (1975) found that ^{14}C fragments were incorporated into fatty acid, glycogen, and amino acid components of the fish.

In a report by NRCC (1978) it was concluded that 2,4-D was not accumulated in major links in food webs, and that 2,4-D residues in shellfish, benthic fauna, and fish reported by Smith and Isom (1967), Whitney et al. (1974), and Coakley et al. (1964) were there as a result of residues in plankton and plant pools. Residues in plants have been observed to persist for 2 to 6 months by Wojtalik et al. (1971). Model ecosystem studies by Isensee (1971) and Metcalf and Sanborn (1975) suggest that at aquatic concentrations of 0.1-0.2 mg/l, 2,4-D accumulation would be observed in algae and daphnids in magnitudes of 1 to 2 orders greater than in water.

Toxicity Data Evaluation

As summarized in the main body of this report, there is no clear evidence available that indicates that 2,4-D is a carcinogen, although considerable debate has been generated on the subject and further study is needed. There is some evidence to suggest that 2,4-D causes a weak teratogenic effect; however, the data present no firm basis for conclusion. Although there are some conflicting results, most reliable tests indicate that 2,4-D is not a mutagen. Insufficient information is available on the toxicity of the various forms of dioxin found in 2,4-D.

The epidemological tests reviewed in this study are inadequate because of small sample size and an inability to factor out the effects of other chemicals to which the workers were exposed. Reports of neurotoxicity are also suspect, due to an inability to factor out exposure to other chemicals. Also, the neurotoxicity reported is difficult to make consistent with the lack of neurotoxic effect from acute exposure in frequent homeowner use for several decades.

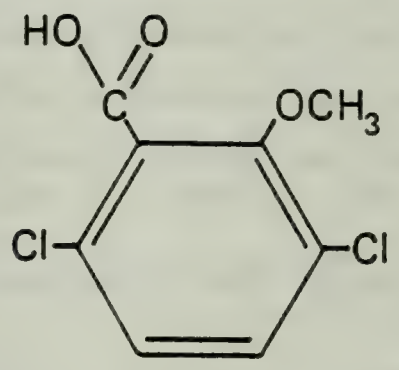
No studies in EPA registration files have been conducted by IBT.

According to the Massachusetts Conservation Law Foundation, EPA identified the following data gaps in registration files: acute toxicity, tumor formation, reproduction, birth defects, neurotoxicity, and metabolism. Acute toxicity data have been submitted.

F. DICAMBA

1. INTRODUCTION

Dicamba is the common name for the herbicide 2-methoxy-3, 6-dichlorobenzoic acid (Velsicol Chemical Corp., 1981) or 3,6-dicloro-o-anisic acid (TRW, 1981; Thomson, 1975), manufactured by Velsicol Chemical Company. It is also known as Banvel®, Banex®, Dianat®, Mediben®, and Mondak® (Thomson, 1976). Banvel 720® and Banvel 520® are formulations that also contain 2,4-D (the DMA salt and the isooctyl ester of 2,4-D, respectively). Dicamba formulations include granules, pellets, an oil-soluble acid, and various water-soluble acids and salts (Velsicol Chemical Corp., 1981). The structure of dicamba is



Other relevant physical and chemical characteristics are presented below in the discussion of the fate of dicamba in soil and water.

2. TOXICITY

Acute Toxicity

Acute toxicity tests show a low order of toxicity for dicamba. Oral LD_{50} values, summarized in Table II-23, range from 1028 mg/kg to 2900 mg/kg for rats, and greater than 4640 mg/kg for mice. Guinea pig and rabbits appear to be more sensitive with LD_{50} values of 566 mg/kg, reported for both by Velsicol Chemical Corporation (1974a).

TABLE II-23

ACUTE ORAL TOXICITY OF DICAMBA

Form	LD_{50} (mg/kg body weight) Rat	Guinea Pig	Rabbit	Mouse	Source
Technical Banvel	1707-2900				Velsicol Chem. Co. (1979)
Banvel, 4 lb/gal DMA	1028-2629				"
Dicamba, DMA salt	2900				Ingle (1962)
Acid	2900 ± 800				Velsicol Chem. Co. (1974); Malina (1973)
Acid	2740-2900			>4640	Velsicol Chem. Co. (publication date not given)
DMA	1028	566	566		Velsicol Chem. Co. (1974)
DMA	1028				Malina (1973)
Dicamba (3,6-dichloro-o-anisic acid)	1040				Thomson (1975-1976)
3,6-dichloro-salicylic acid	1440				Zick and Castro (1966)

Although several methoxy derivatives of dicamba are less toxic than the parent compound, 3,6-dichlorosalicylic acid, the major decomposition product, is as toxic as the parent compound. Its oral LD_{50} is 1440 mg/kg in rats (Zick and Castro, 1966).

Velsicol Chemical Corporation (1974a) found the dermal LD_{50} value to be greater than 2000 mg/kg when technical Banvel and Banvel DMA salt (4 lb/gal) were administered to the skin of rabbits. In regard to inhalation toxicity, they reported that the concentration of Banvel DMA in air necessary to give a 50% probability of lethality in 4 hours was greater than 200 mg/l.

In a study by Edson and Sanderson (1965), intraperitoneal administration of technical grade dicamba to the rat resulted in an LD_{50} value of 80 mg/kg. When it was administered subcutaneously, the LD_{50} was 1000 mg/kg.

TRW (1981) states that some formulations are extremely corrosive and irritating to the eyes, citing a Velsicol Chemical Corporation Bulletin (1974a). No other information was provided. A low-grade irritation of the eye of the rabbit was noted when 0.1 ml of a 4 lb/gallon concentrate of dicamba as the diethylamine salt (89.7%) was administered. This disappeared rapidly and no injury to the cornea or iris was observed (Velsicol Chemical Corporation, 1974b). When the dimethylamine salt was applied to the eyes of the rabbits as a 0.2% or 2% aqueous solution in single or repeated doses for a week, no irritation or injury was noted (Velsicol Chemical Corp., 1974b).

Subchronic Toxicity

Dicamba has a low order of toxicity in subchronic studies except at high doses or long exposures. Dicamba as the amine salt was fed to rats for 13 weeks at dietary concentrations of 100, 500, 800, and 1000 ppm. After 7 weeks there were no deaths, the pathology was negative, and growth rates and food consumption were normal. After 13 weeks, there still were no detectable effects at 100 or 500 ppm. However, at

800 ppm slight liver pathology was noted, and at 1000 ppm moderate histopathological effects on the liver and kidney were observed (Velsicol Chemical Corporation, 1974b). No significant toxic effects were observed on rats fed for three weeks on diets containing 658 ppm to 23,500 ppm Banvel D, as reported by EPA (1975).

Edson and Sanderson (1965) conducted a feeding study using male Wistar rats. For 15 weeks, the rats were fed diets containing 31.6, 100, 316, 1000, and 3162 ppm dicamba. At 1000 and 3162 ppm, they noted a slight but statistically insignificant increase in the liver-to-body weight ratio. They estimated that 316 ppm dicamba (equivalent to 19 mg/kg/day was the dose at which no adverse effect was seen.

Kudzina and Golovan (1972) concluded that there were no adverse signs of toxicity in rats and rabbits fed 0.075 or 0.75 mg/kg/day for a period of 6 months. The authors noted toxic effects in the animals fed 7.5 mg/kg/day, although these effects were unspecified.

A bulletin from Velsicol (1974a) stated that a mild irritation resulted from administration of undiluted dicamba (dimethylamine salt) to the skin of rabbits and rats for 2 weeks. No irritation was noted after the dicamba was diluted 1:40 in water and applied to the skin for 30 days.

Mammalian Metabolism

Dicamba appears to be rapidly excreted from the body, as would be expected as a consequence of its high water solubility. Rapid excretion in urine was observed in a feeding study on dogs in which 88% of the labeled dose was excreted unchanged and 12% was excreted in conjugation with glycine. (Velsicol, 1974a). When labeled dicamba was administered orally to rats, 93%-99% of the label was excreted in the urine as unchanged dicamba, with small amounts in the form of glucuronide congugates. From 1% to 4.5% of the labeled material was found in feces (Tye and Engel, 1967).

Oral administration of 20,000 ppm labeled dicamba to a heifer resulted in the urinary excretion of unchanged dicamba, with a hydrolysis product 3,6-dichlorosalicylic acid also detected (Edson and Sanderson, 1965). Only 73% of administered radioactivity was excreted after 7 days in the urine of a Holstein cow fed 5 ppm dicamba for 4 days (St. John and Lisk, 1969).

Special Studies

Carcinogenicity No evidence of tumor induction was observed in a dog study in which purebred beagle dogs were fed dicamba at dietary levels of 0, 5, 25, and 50 ppm for two years. In another study, no evidence of carcinogenicity was seen when male and female Sprague-Dawley rats were fed 0, 5, 50, 100, 250, and 500 ppm dicamba (USEPA, 1975). In both of the above studies, no adverse effects were observed in survival, food consumption, body weight, organ weight, hematology or histology. Personal communication with Dr. David Whitacre (9/8/83) of Velsicol Chemical Corporation indicates that the rat study was conducted at least 15 years ago and that a new 2-year rat study, using an updated laboratory protocol, is nearing completion. Results will be available in the late spring of 1984.

Teratogenicty/Reproduction A review by EPA (1975) cities two studies which show no reproductive toxicity. In one study, Charles River-CD rats were fed 206 ppm dicamba. No reproductive toxicity was observed over a period of three generations. Similar results were found when 500 ppm dicamba was included in the diets of Sprague-Dawley rats (USEPA, 1975).

Dunachie and Fletcher (1970) injected 10-400 ppm dicamba into chicken eggs and found a 38% reduction in hatching at the highest dose. It should be noted that there was a great deal of variability in the percentage of hatching, and that no clear dose response was evident for any of the 25 herbicides tested. Furthermore, the absence of a physiologic maternal-fetal relationship during incubation makes this test system highly questionable for assessing potential reproductive or teratogenic hazards in humans.

Mutagenicity As summarized in Table II-24, available studies show no mutagenic effect. One of the studies showed mixed results (Anderson et al., 1972). This study found that dicamba showed a statistically significant ($p = 0.05$) increase in induction of rII mutants of the T_4 bacteriophage. However, the herbicide did not result in a "marked increase" as compared to other substances, and no increase in mutations was found in test systems using eight histidine-requiring mutants of Salmonella typhimurium. Eisenbeis et al. (1981) tested dicamba alone and dicamba in combination with atrazine. No increase in mutations over controls was found in either test.

Other Information Related to Chronic Toxicity Bontoyan et al. (1979) screened a variety of technical and commercial pesticide formulations for the presence of nitrosamine contaminants. No such contaminants were found in dicamba samples, although it should be noted that the level of detection was only 1 ppm.

Summary An evaluation of important toxicity data is provided at the end of this chapter.

3. MOBILITY AND PERSISTENCE

Fate in Soil

Dicamba has been found to be one of the most mobile of all herbicides (TRW, 1981; USDA, 1973; Malina, 1973). In its pure form, dicamba has a moderately low solubility (0.65 gm/100 ml at 25°C), but as the more commonly used dimethylamine salt, it has a very high water-solubility of 72 g/100 ml at 25°C. Sodium and potassium salts are also highly water-soluble (USDA, 1973). Friesan (1965) found that when an unspecified amount of dicamba was applied to a sandy loam and eluted with 5 cm of water, the herbicide reached a depth of 15 cm in 1 hour. Dicamba was found to be the most mobile of 40 pesticides tested, with a mobility value of 0.96 (the range for the 40 pesticides was 0 to 0.96) (Helling, 1971). In a study of 28 herbicides, dicamba was found to be more mobile than all but one (2,3,6-trichlorobenzoic acid) (Harris, 1967). After 63 weeks, dicamba had reached a depth of 68 cm in a sandy loam (EPA, 1975; no primary source given). A review of the literature by

TABLE II-24

MUTAGENICITY TESTS: DICAMBA

Test System	Result	Comments	References
Saccharomyces cerevisiae D3			
Mitotic recombination assay	-		Poole et al. (1972)
Ames/Salmonella	-		Poole et al. (1972)
	-	5 µg/plate	Anderson et al. (1972)
	-		Eisenbeis et al. (1981)
	-	Strains TA-98, TA-100	Moriya et al (1983)
Escherichia coli WP2	-		Poole et al. (1972)
	-		Moriya et al. (1983)

Velsicol (1981) states that many studies indicate that dicamba will move vertically and that it has been shown to move with the flow of water (no primary sources given except Naishtein et al., 1981). Dicamba can also move up vertically into the root zone as evaporation draws soil moisture upward (Harris, 1963).

The high mobility of dicamba indicates that dicamba does not adsorb strongly to soil particles. Dicamba does not adsorb to illite clays (Burnside and Lavy, 1966). Kaolinite clays can adsorb some dicamba because of their anionic exchange capacity (dicamba behaves as an anion between pH 4.1 and pH 9.4) (Burnside and Lavy, 1966).

A number of studies have shown that dicamba is adsorbed on organic matter (Grover, 1977; Khan, 1973; Stewart and Gaul, 1977). Corbin et al. (1971) showed that dicamba is more strongly adsorbed at low pH. Velsicol (1981) also states that the adsorption of dicamba is negatively correlated with pH.

After summarizing both internal and publicly available studies, Velsicol (1981) states, "In summary, dicamba is mobile in soil. High organic matter or low pH may retard movement of the substance but certainly does not eliminate it. Therefore, vertical soil mobility will account for some portion of the loss of dicamba from surface soils."

The major soil degradation product, 3,6-dichlorosalicylic acid, is more readily adsorbed than the parent compound, with at least 30% of the applied material bound to soil colloids in a variety of soil types (Smith, 1974).

In regard to runoff, TRW (1981) concludes that this route of loss is not likely to be significant because dicamba salts are so highly water-soluble and quickly move downward in the soil. Runoff should result only in cases where rainfall occurs very soon after application and is sufficiently heavy to result in significant lateral as well as vertical movement. Similar conclusions were reached by Velsicol (1981). Trichell, et al. (1968) measured dicamba concentrations from two clay loam plots

(3% slope), one with sod and one that was fallow. After 24 hours, the concentrations of dicamba in runoff water were 4.81 ppb and 1.60 ppb from the sod and fallow plots, respectively. After 4 months, these concentrations dropped to 0 and 0.018 ppb.

The persistence of dicamba is difficult to assess because of the overriding importance of the amount of water that moves through the soil. Phytotoxic levels have been reported to persist from as little as 30 days (Burnside and Lavy, 1966), to over 1 year (Dowler et al., 1968). Arthur D. Little, Inc., (1979) reviewed the literature to determine the persistence of dicamba in sandy loam soil (a common soil in Massachusetts) and found four studies indicating persistence ranging from 2 months to over a year. Velsicol (1981) states that dicamba will have a half-life of less than 30 days under most conditions. Altom and Stritzke (1973) showed that the dimethylamine salt of dicamba had a half-life of 17-32 days when applied to forest and grassland soils at a concentration of 2.47 ppm. A faster dissipation rate was observed by Stewart and Gaul (1977), who applied an amine salt of dicamba to a silty loam at rates up to 4.5 kg a.i./ha. After 42 days, 5% of the dicamba remained. Audus (1964) and Cain (1966) found that within 10 months after application of up to 6 lbs/acre of dicamba to an unspecified soil, all had disappeared below detection limits in the top 24 inches of soil. Scifres and Allen (1973) state that at application rates of 1.12 kg/ha or less, dicamba should not persist longer than one growing season when applied in spring.

In a laboratory study, Smith (1974) found that over 50% of dicamba in moist, non-sterile heavy clay was lost in 4 weeks. The major degradation product, 3,6-dichlorosalicylic acid, increased as dicamba degraded, but then decreased to non-detectable amounts in 9 weeks.

Numerous studies suggest that dicamba is stable to chemical hydrolysis and that degradation is predominantly microbial (Smith, 1974). Smith and Cullimore (1975) showed that while dicamba did not dissipate in sterilized soils at 15°C, it did degrade significantly at the same temperature in unsterilized soils.

The factors that affect microbial degradation of dicamba were reviewed by Velsicol (1981). They found the most important factor to be a healthy microbial population. The degradation of dicamba has been shown to increase with conditions that promote microbial growth, namely organic matter, moisture, and high temperatures (Arthur D. Little, Inc., 1979). Addition of bacterial nutrient broth has been observed to accelerate degradation (McClure, 1970).

A number of studies have also shown the pH of the soil to be an important factor. In soils with a high percentage of organic matter, the optimum pH for degradation of dicamba was found to be 5.3 (Corbin and Upchurch, 1967). Velsicol (1981) states that dicamba was found to be considerably more persistent at pH 7.5 than at lower pH's. Others have found that degradation of dicamba increases with decreasing pH (Swanson, 1969; Parker and Hodgeson, 1966).

Harger (1975) and Smith (1973, 1974) both state that during degradation, dicamba is decarboxylated in the soil and the ring is opened. The only degradation products documented by Smith (1974) were 3,6-dichlorosalicylic acid and CO_2. Velsicol (1981) states that in addition to these two products, unspecified "tightly bound materials" are produced.

Some loss of dicamba can be expected to occur from volatilization, although the total amounts are probably not significant. After incorporation of dicamba into soil, vapors were found to be toxic to beans in a study by Gentner (1964) (no time specified). In another bioassay, volatilization of dicamba from corn leaves was detected for 3 days after the corn leaves had been treated (Behrens and Lueschen, 1979). These same authors, however, found very little loss of dicamba during an 8-week period from autoclaved soils at 35°C and under humidities ranging from 0% to 100%.

Residues and Persistence in Water

Residues of dicamba in streams have been found to be low. Bonneville (1981, no primary source given) described a 3-year study in western

Washington in which 0.5 lb/acre of dicamba was applied by helicopter to a transmission line right-of-way. In the first year, an (unplanned) direct application was also made to the stream. In that year, 8 ppb, 1 ppb, and approximately 3 ppb were detected 30 hours, 48 hours, and 2 weeks, respectively, after application. In the second year, at an application rate of 1 lb/acre, no dicamba residues were found. In the last year, at the same application rate, dicamba residues peaked at 3 ppb after 4 hours and then diminished to non-detectable levels after 4 days.

For 14 months, Norris and Montgomery (1975) sampled the water from a stream adjacent to a forest application of 1.12 kg/ha of dicamba. No residues were detected beyond 11 days after application. Residual levels peaked at 37 ppb in about 5.2 hrs, then declined to background levels in 37.5 hrs.

Very little is known concerning the routes of loss in water. Photodecomposition of dicamba is known to occur (USDA, 1973), although the total amounts lost by this route are probably not significant. There is some evidence to suggest that dicamba is removed by adsorption to stream sediments (Norris and Montgomery, 1975) and uptake by aquatic vegetation (USDA, 1973). Velsicol (1981) states that dicamba has been directly applied to standing water and found to dissipate rapidly (no data provided).

Very little information is available on the degradation products of dicamba in water. In the laboratory, Chirchirillo (1966) and Allen and Scifres (1971) both report the formation of 3,6-dichlorosalicylic acid. Yu et al. (1975) detected small amounts of 5-hydroxydicamba. Neither metabolite was found in water in the field after forest spraying (Norris and Montgomery, 1975) or after dry ditchbank treatment (Salman et al., 1972).

Indicators of Potential Ground Water Contamination

Table II-25 provides information on parameters associated with the mobility of dicamba. These parameters, and their associated

thresholds, have been suggested by EPA for use in assessing the potential for pesticide contamination of ground water. A discussion of these parameters and thresholds, and the methods for arriving at designated values for individual herbicides, is presented in the main body of the report as part of the discussion of the fate of herbicides in the environment.

TABLE II-25

INDICATORS OF POTENTIAL GROUND WATER CONTAMINATION: DICAMBA

Indicator	Value for Dicamba	Threshold
Solubility	6500 ppm (technical material) at 20°C;720,000 ppm (dimethyl salt)	>30 ppm
K_{oc}	<150**	<300-500
Speciation at pH 5	Anionic	Anionic (negatively charged)
Hydrolysis half-life	ND*	>6 months
Photolysis half-life	ND*	>3 days
Vapor pressure	3.41×10^{-5} mm Hg at 25°C	$<10^{-2}$mm Hg

* ND = no data.
** Based and K_d<1 (Velsicol).

4. TOXICITY TO NON-TARGET ORGANISMS

Birds

Information from the U.S. Forest Service (1974) and Velsicol Chemical Corporation (1974a), summarized in Table II-26, showed that dicamba can be considered non-toxic to birds. LC_{50} values ranged from 673 to 2000 mg/kg for domestic hens, mallard ducks, bobwhite quail, and pheasants.

TABLE II-26

THE EFFECT OF DICAMBA ON BIRDS

Form	Species	Test	Results	Source
Technical Banvel (86.8% a.i.)	Mallard ducks	Acute oral LC_{50}	2000 mg/kg	Velsicol Chemical Corp. (1974a
	Mallard ducks	8-day dietary LC_{50}	>10,000 ppm	"
	Bobwhite quail	8-day dietary LC_{50}	>10,000 ppm	"
Banvel XP (10% a.i.)	Mallard ducks	8-day dietary LC_{50}	>10,000 ppm	EPA (no date, [c])
	Bobwhite quail	8-day dietary LC_{50}	>10,000 ppm	"
Banvel-310, 4 lb/gal DMA	Mallard ducks	8-day dietary LC_{50}	4640 ppm	Velsicol Chemical Corp. (1974a
	Bobwhite quail	8-day dietary LC_{50}	4640 ppm	"
Technical Banvel-310	Mallard ducks	Acute oral LC_{50}	2000 mg/kg	EPA (no date, [c])
	Pheasants	LC_{50}	673 mg/kg	U.S. Forest Service (1974)
	Domestic hens	LC_{50}	800 mg/kg	"

Fish

Table II-27 shows that tests with rainbow trout, bluegills, spot, and juvenile Coho salmon indicate that 96-hr LC_{50} values range from 23-130 ppm. A report by Arthur D. Little, Inc. (1979) considered dicamba "moderately toxic" to fish, depending on the species. One study by Mills and Lowe (Gulfbreeze Lab, unpublished data [a]) reported a 48-hour LC_{50} value for spot (Leiostomus xanthurus) of 1 ppm.

Lower Aquatic Organisms

Conflicting data are available on the effect of dicamba on lower aquatic organisms. As shown in Table II-28, studies by Velsicol Chemical Corporation (1974a) and Sanders (1969, 1970) showed LC_{50} test results of greater than 100 ppm for a variety of aquatic crustaceans. However, studies by Sanders (1969) and the U.S. Department of the Interior, FWPCA (1968) reported toxicity levels for a marine amphipod (Gammarus lacustris) of between 5.8 and 10.0 ppm. Mills and Lowe (Gulbreeze Laboratory, unpublished data [a]) reported an EC_{50} (for 48-hour exposure) as 1.0 ppm. The same study showed oysters to have a 50% reduction in shell growth after exposure to 5 ppm for 96 hours.

Livestock

Malina (1973) reports that dairy cattle given food with residues from 10 to 400 ppm of dicamba showed no adverse reactions. Assays of the milk showed no residues at the lower levels, and residues not exceeding 0.15 ppm appeared in the milk after the cow was fed 400 ppm for 9 days. Vital organ samples showed no trace of dicamba when cattle grazed for 30 days on pasture treated with 5 and 10 lb Banvel/acre.

Bees

Available studies present conflicting results in regard to the toxicity of dicamba to bees. A study by Atkins et al. (1973) reported that exposures of 90.65 µg/bee produced a mortality of 2.58% after exposure for 96 hours. Edson and Sanderson (1965) reported an LD_{50} of 3.6 µg/bee when administered orally in a 20% solution.

TABLE II-27

THE EFFECT OF DICAMBA ON FISH

Form	Species	Test	Results	Source
Technical Banvel	Bluegill sunfish	96-hr LC_{50}	135 mg/l	Velsicol Chemical Corp. (1974a)
	Rainbow trout	96-hr LC_{50}	135 mg/l	"
	Sheephead minnow	96-hr LC_{50}	>180 mg/l	"
Banvel, 4 lb/gal DMA	Bluegill sunfish	96-hr LC_{50}	>1000 mg/l	"
Dicamba, DMA salt	Bluegill sunfish	48-hr LC_{50}	130 ppm	"
	Rainbow trout	48-hr LC_{50}	35 ppm	Pimentel (1971)
	Rainbow trout	24-hr LC_{50}	35 ppm	U.S. Forest Service (1974)
	Rainbow trout	96-hr LC_{50}	28 ppm	"
	Bluegill	24-hr LC_{50}	130 ppm	"
	Bluegill	96-hr LC_{50}	23 ppm	"
	Coho salmon	24-hr TL_{50}	151 ppm	"
	Coho salmon	48-hr TL_{50}	121 ppm	"
Dimethylamine salt	Carp	24-hr LC_{50}	659 ppm	"
Dicamba	Spot (Leiostomus xanthurus)	48-hr LC_{50}	1 ppm	Mills and Lowe (Gulfbreeze Labs, unpublished data [b])

TABLE II-28

EFFECTS OF DICAMBA ON LOWER AQUATIC ORGANISMS

Species	Test[1]	Result	Source
Daphnia	48-hr LC_{50}	111 mg/l	Velsicol Chemical Corp. (1974a)
Grass shrimp	96-hr LC_{50}	>100 mg/l	"
Fiddler crab	96-hr LC_{50}	>180 mg/l	"
Sowbug	48-hr TL_{50}	>100 ppm	Sanders (1970)
Crayfish	48-hr TL_{50}	>100 ppm	"
Seed shrimp	48-hr TL_{50}	>100 ppm	"
Brown shrimp	48-hr LC_{50}	1 ppm	Mills and Lowe (Gulfbreeze Labs, unpublished data)
Gammarus lacustris	24-hr LC_{50}	10 ppm	Sanders (1969)
"	48-hr LC_{50}	5.8 ppm	"
			USDI FWPCA (1968)
"	96-hr LC_{50}	3.9 ppm	Sanders (1969)
Cypridosis vidua	48-hr LC_{50}	>100 ppm	Sanders (1970)
Palaemonetes kakiakensis	48-hr LC_{50}	>100 ppm	"
Orconectes nais	48-hr LC_{50}	>100 ppm	"
Crassostrea virginica	96-hr LC_{50}	5.0 ppm	Mills and Lowe (Gulfbreeze Labs, unpublished data [b])
Asellus brevicandus	48-hr LC_{50}	>100 ppm	Sanders (1970)

[1] Technical Banvel used in all studies.

Other studies by Morton et al. (1972) and Atkins et al. (1975), in which bees were fed or dusted with dicamba at suggested field concentrations, produced no significant mortality.

Soil Microorganisms

User handbook information supplied by the manufacturer stated that soil microorganisms were not harmed by dicamba (Thomson, 1975). Cain (1966) and Andus (1964) noted that Bacillus cereus var. mycoides was capable of decarboxylating dicamba.

Bioaccumulation

Dicamba is readily soluble in water, and a report by TRW (1981) concluded that there was no evidence to suggest that it was magnified in the food chain. No data were provided.

Toxicity Data Evaluation

Data on the carcinogenic, mutagenic, and teratogenic effects of dicamba are inadequate to enable conclusions to be drawn. Additional data need to be generated in regard to all these effects. (Although, according to the registration standard, one teratogenicity study using female rabbits has been reviewed and found to show no positive effects.)

A number of studies on dicamba were conducted by IBT. Of the mutagenicity tests, two using mice were found to be invalid, and no response has yet been made by Velsicol regarding their replacement. One mutagenicity test using bacteria was also found to be invalid. Velsicol has notified EPA that it does not intend to replace that test. One study in EPA files on the mutagenicity of dicamba was not conducted by IBT.

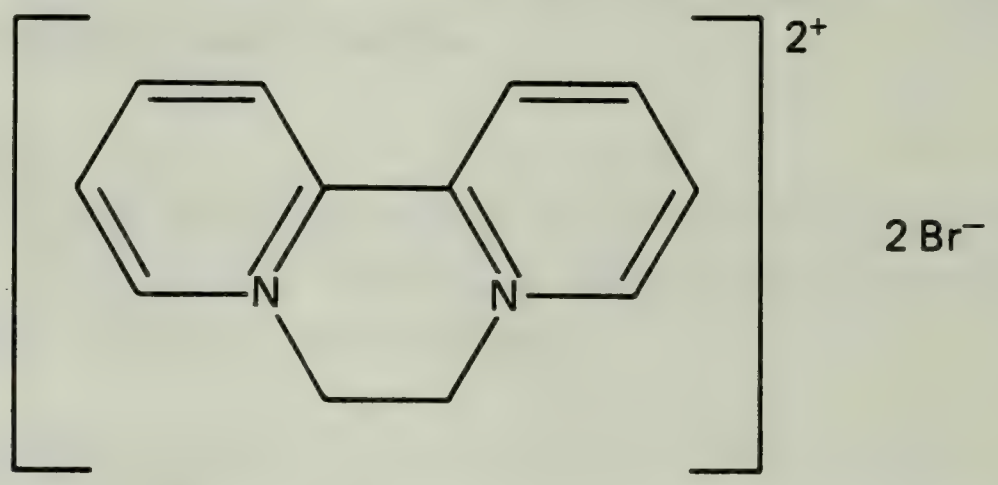
A teratogenicity and reproduction study done by IBT were also found to be invalid. The former has been replaced, and the latter has been determined to be not required by EPA. Several teratogenicity and reproduction studies conducted by other laboratories already exist in EPA files. Finally, a chronic/carcinogenicity study is currently being replaced by Velsicol.

In its registration standard review, EPA decided the following information is required to be submitted before products containing dicamba can be reregistered: 90-day feeding study on nonrodents; 21-day dermal study; 90-day inhalation study; chronic oral study on 2 species; oncogenicity on 2 species; gene mutation; chromosomal aberration; and other mechanisms of mutation.

G. DIQUAT

1. INTRODUCTION

Diquat is the common name for the herbicide 6,7-dihydrodipyridol pyrazidinium dibromide, produced by Chevron Chemical Company. It is also known as diquat dibromide, Reglone®, Aquacide®, Dextrone®, and Weedrite®. (Thomson, 1975; Ouellette and King, 1977). Diquat is a quaternary salt of 4,4-dipyridyl, with the following chemical structure:



Relevant chemical and physical properties are presented below in the discussion of the fate of diquat in soil and water.

2. TOXICITY

Acute Toxicity

Toxicological information indicates that diquat can be considered moderately toxic. Thomson (1975) reports an LD_{50} of 231 mg/kg (no test organism given). Manzo (1979) reports an LD_{50} for rats to be 400-440 mg/kg. A Material Information Bulletin by Chevron Chemical Company (1982) states that diquat may be toxic to humans if swallowed, and that the oral LD_{50} for rats is 600 mg/kg for females and 810 mg/kg for males. Calderbank (1968) reports oral LD_{50} values ranging from 30 to 400 mg/kg for cattle, mice, dogs, rats, and chickens. Cattle were the most sensitive and chickens were the least sensitive.

In regard to dermal toxicity, the Material Information Bulletin by Chevron Chemical Company (1982) states that the acute dermal LD_{50} for rabbits is 260 mg/kg (male) and 315 mg/kg (female).

Both the label and the Material Information Bulletin by Chevron Chemical Company (1982) state that contact with the skin may cause severe irritation to skin, and that diquat may be fatal if absorbed through skin. No data is cited. These two information sources also state that diquat may be fatal if inhaled. The Material Information Bulletin also lists an acute inhalation LC_{50} for rats (exposed for 1 hour) to be less than 6.1 mg/l, and that "breathing spray mist may cause nasal, throat, and respiratory tract irritations."

Subacute/Subchronic Toxicity

Black et al. (1966) and Calderbank (1968) report no observable adverse effects on several animals tested by feeding silage and hay containing up to 20 mg/kg diquat for 90 days. Howe and Wright (1965) noted no toxic symptoms for sheep and calves given drinking water treated with 20 mg/l diquat for 30 days.

Mammalian Metabolism

Stevens and Walley (1966) fed ^{14}C-diquat (5 to 20 mg/kg) in single oral doses to lactating cattle and found radioactivity largely as metabolites in urine (2.6%), in milk (0.02%), and the remainder in feces. Daniel and Gage (1966) and Black et al. (1966) demonstrated that metabolites in rats and sheep originated from microbial breakdown in the gut.

Work by Stevens and Walley (1966), Black et al. (1966), Calderbank (1968), and Litchfield et al. (1973) suggests that the milk and meat of animals injesting "normal" levels of diquat would be free of contamination due to the rapid excretion from the body (largely as metabolites). Daniel and Gage (1966) report that diquat is a highly soluble divalent cation that is stable and not metabolized to any significant extent after administration to animals.

Special Studies

Carcinogenicity No data were located regarding the carcinogenic potential of diquat.

Teratogenicity/Reproduction Limited data suggest a possible teratogenic effect. CFLP mice were given either repeated administrations (4) of a low dose (2.7 mg/kg) or a single administration of a higher dose (11.0 mg/kg). The repeated administration of the lower dose resulted in a retardation in weight gain. At both doses, changes in the skull, vertebrae, sternum, and limbs were observed. The number of dead or resorbed embryos was increased 9% with the single administration of the higher dose, and 11% with repeated administration of the lower dose.

Using the egg injection technique, Dunachie and Fletcher (1970) noted an embryotoxic effect after administration of 10 ppm. Hatching was reduced 93%, compared to controls. It should be noted that a great deal of variability in the percentage of hatching was seen and no clear dose response was evident for any of the tested herbicides. Furthermore, the absence of physiologic maternal-fetal relationships during incubation make this test system of questionable value in assessing potential teratogenic hazard to humans.

Mutagenicity As shown in Table II-29, available studies indicate that diquat is not a mutagen.

Summary An evaluation of important toxicity data is provided at the end of this chapter.

TABLE II-29

MUTAGENICITY TESTS: DIQUAT

Test System	Result	Comments	Sources
in vivo mouse bone marrow	-	90 mg/kg orally of Reglone® (contains 20% diquat) or 7.3 mg/kg i.p.	Selypes et al. (1980)
Mouse dominant lethal	-	76 nmoles/kg i.p.	Pasi et al. (1974)
Aspergillus nidulans	(+)*	400-600 µg/plate	Benigni et al. (1979)
Streptomyces coelicolor	-		"
Saccharomyces cerevisiae Mitotic gene conversion	-		Fahrig (1974)
Saccharomyces marcescens	-		"
Ames/Salmonella	-	1000 µg/plate	Benigni et al. (1979)
	-	5 µl/plate	Anderson et al. (1972)
	-	0.7 µg/plate ± liver or plant enzyme homogenate	Wildeman and Nazar (1982)
	-		Moriya et al. (1983)
Thilly's forward Ames	+	0.1-10 µg/plate	Benigni et al. (1979)

TABLE II-29 (CONTINUED)

MUTAGENICITY TESTS: DIQUAT

Test System	Result	Comments	Sources
Escherichia coli WP2	-		Moriya et al. (1983)
	+	2.2 nmoles/ml	Probst et al. (1981)
rII mutants of T_4 bacteriophage	-	20 µg	Anderson et al. (1972)
Unscheduled DNA synthesis			
Human lymphocytes	-	500 µg/ml	Rocchi et al. (1980)
Human heteroploid cells	+	20-1000 µg/plate	Benigni et al. (1979)
Rat hepatocytes	-		Probst et al. (1981)
Pelargonium zonale	+		Pohlheim and Gunther (1977)

* Parentheses indicate uncertainty.

3. MOBILITY AND PERSISTENCE

Fate in Soil

Limited information is available on the fate of diquat in soil and water. Most of the information presented below comes from a review by Simsiman et al. (1976), who presented little detail regarding the reviewed studies.

Given these qualifications, however, it appears that diquat has low mobility, since it is adsorbed strongly by clay and organic matter in the soil. The bypyridyl herbicides (including diquat) were retained in the upper 0.01 cm of soil after elution with water equivalent to 11 months of natural rainfall, even in sandy soil (Coats et al., 1966). It has been estimated that about 10,000 kg/ha of diquat would be needed to saturate the capacity of a sandy clay loam soil to adsorb diquat (Knight and Tolimson, 1967).

Diquat is very strongly adsorbed to clay particles and can occupy the entire cation exchange capacity of montmorillonite and kaolinite, and from 30%-90% of the capacity of vermiculite (Weber et al., 1965; Weber and Weed, 1968; Weed and Weber, 1969; Dixon et al., 1970). Adsorption onto clay appears to be independent of pH and temperature, and equilibirium (adsorption/desorption) is reached after an hour (Harris and Warren, 1964; Weber et al., 1965; Coats et al., 1966). The amount of montmorillonite in the soil is particularly important in determining both persistence and mobility. Because diquat molecules can occupy the spaces between the layers of montmorillonite, diquat is strongly retained once it is adsorbed. Only 5%-10% of the bound diquat can be released from montmorillonite by competing cations (Al^{+++}, Ba^{++}, Ca^{++}, Mg^{++}), while 80%-90% can be released from kaolinite and vermiculite (Weber et al., 1965; Weber and Weed, 1968; Weed and Weber, 1969). Diquat can be desorbed from montmorillonite, however, by similar organic cations, such as paraquat. Paraquat displaces 50%-90% of the bound diquat on montmorillonite and 100% on kaolinite (Weber et al., 1965; Weber and Weed, 1968).

Diquat is also readily adsorbed onto organic matter such as humic substances (Damanakis et al., 1979), organo-clay complexes (Khan, 1973), and peat, muck, and organic soils (Harris and Warren, 1964; Scott and Weber, 1967; Tucker et al., 1967). Binding to organic matter appears to be weaker than binding to clay. Diquat was displaced more readily from organic matter than from clay when exposed to solutions of competing inorganic cations (Simsiman, et al. 1976).

Although no data is presented, Simsiman et al. (1976) suggests that diquat may move by erosion and runoff because of its tendency to accumulate in the upper layer of soil.

Microbial degradation of diquat has been demonstrated by a number of investigators (Baldwin et al., 1966; Tu, 1966; Slodki and Wickerham, 1966; and others). No degradation of diquat occurred under sterile conditions in a study by Weber and Coble (1968) using labeled diquat and soil in nutrient solution cultures. Under unsterile conditions, decomposition of diquat released labeled CO_2. The metabolic pathway of diquat has yet to be determined (Simsiman, et al. 1976).

It should be noted that diquat adsorbed to montmorillonite is not available for microbial attack (Weber and Coble, 1968). Montmorillonite may thereby increase the persistence of diquat. Unfortunately, no information regarding the half-lives of diquat in various soils is available.

Photodegradation may be an important route of loss of diquat on the surface of soils and on plant surfaces. Ultraviolet light has been found to degrade diquat rapidly (Slade, 1965 and 1966; Coats et al., 1966; Funderbank et al., 1966; Slade and Smith, 1967).

Simsiman et al. (1976) summarizes a review of the information on the fate of diquat in the soil with the following statement: "The avenues of loss of diquat in soils seem to be through photochemical and microbial degradation. Since adsorption tends to slow the rate of these

processes, it is likely that diquat will accumulate in soils, particularly those containing montmorillonite."

Persistence in Water

Diquat has been observed to disappear rapidly from natural waters (Coats et al., 1964; Grzenda et al., 1966; Frank and Comes, 1967; Yeo, 1967; Hiltibrand et al., 1972). Diquat residues were found to be undetectable after 8 days in a study by Coats et al. (1964), and after 4 days in a study by Frank and Comes (1967). Yeo (1967) found only trace residues after 12 days when diquat had been applied at the rate of 0.125 to 2.5 µg/ml.

It appears that diquat dissipates by adsorption to sediments and suspended particulate matter and uptake by aquatic plants (Davies, 1964; Grzenda et al., 1966; Frank and Comes, 1967; Yeo, 1967; Hiltibrand et al., 1972; Simsiman and Chesters, 1975). Absorption of diquat by aquatic plants may be a significant route of loss, as suggested by the ability of aquatic plants to concentrate the herbicide by a factor of 50 after 4 days from an initial concentration of 0.5 µg/l (Newman and Way, 1966). In another study, an initial concentration of 0.62 µg/l disappeared from pond water in four days, but was then detected after 24 days in sediments, increasing in the sediments to a concentration of 37 µg/g after 56 days. (Frank and Comes, 1967). Simsiman et al. (1976) suggests that a significant portion of the herbicide is absorbed by the weeds, and that decomposition of those weeds is necessary before the diquat becomes concentrated in the sediments.

After being adsorbed by the sediment, it appears that diquat persists for a considerable time. Four years after 0.3 kg/ha was applied to water bodies, concentrations of diquat up to 1.7 µg/g were found in the sediment (Beasley, 1966).

Indicators of Potential Ground Water Contamination

Table II-30 provides information on parameters associated with the mobility of diquat. These parameters, and their associated thresholds, have been suggested by EPA for use in assessing the potential for pesticide contamination of ground water. A discussion of these parameters and thresholds, and the methods for arriving at designated values for individual herbicides, is presented in the main body of the report as part of the discussion of the fate of herbicides in the environment.

TABLE II-30

INDICATORS OF POTENTIAL GROUND WATER CONTAMINATION: DIQUAT

Indicator	Value for Diquat	Threshold
Solubility	700,000 ppm at 20°C	>30 ppm
K_{oc}	ND*	<300-500
Speciation at pH 5	Cationic (positively charged)	Anionic (negatively charged)
Hydrolysis half-life	ND*	>6 months
Photolysis half-life	ND*	>3 days
Vapor pressure	Non-volatile	$<10^{-2}$mm Hg

* ND = no data.

4. TOXICITY TO NON-TARGET ORGANISMS

Birds

The limited data available suggest that diquat is not toxic to test birds. Heath et al. (1972) and Hill et al. (1975) report LC_{50} values that range from 1346 to >5000 ppm for bobwhite quail, Japanese quail, pheasants, and mallard ducks. Tucker and Crabtree (1970) report the LD_{50} for mallards to be 564 mg/kg.

Fish

The data shown in Table II-31 indicate that diquat is somewhat toxic to a number of species of fish. The 48-hr TL_{50} values range from 11.7 mg/l for lake emerald shiners to 220 mg/l for fathead minnows.

Because diquat is frequently used for aquatic weed control, a number of studies have been done on the effect on fish under field conditions. Gilderhus (1967) found that when diquat was added at a rate of 0.5 to 4.0 mg/l, there was some mortality of mosquito fish; other fish were not harmed by these concentrations. The same study showed that no mortality occurred among fingerlings and adult bluegills exposed to 1 and 3 mg/l in artificial ponds. Blackburn and Weldon (1963) observed no toxicity to fish over a 3-year test using diquat as an aquatic herbicide in concentrations of 2.5 mg/l or less. However, Hiltibran (1967) reports that bluegill fry, lake chubsucker, and small-mouth bass died within 1 to 4 days of exposure to 1.3 to 2.5 mg/l of diquat.

Lower Aquatic Organisms

Table II-32 summarizes available data, most of which resulted from a study by Wilson and Bond (1969). This study concluded that amphipods were very sensitive to diquat, while other invertebrates were able to tolerate higher levels.

Livestock

Cattle may be somewhat sensitive to diquat. Calderbank (1968) states that the LD_{50} value for cattle is 30 mg/kg. Thomson (1976) states that diquat should not be used in the drinking water of animals. On the other hand, Black et al. (1966) and Calderbank (1968) found no adverse effects and no detectable residues in milk or tissue. Also, Howe and Wright (1965) observed no toxic symptoms when sheep and calves were fed water with 20 mg/l of diquat for 30 days.

Bioaccumulation

Gilderhus (1967) and Calderbank (1968) found that the accumulation of diquat in the tissues and organs of fish is negligible except in the digestive tract. Hiltibran et al. (1972) and Beasley (1966) suggest that

TABLE II-31

TOXICITY OF DIQUAT TO FISH

Species	Test	Result (mg/l)	Source
Chinook salmon	24-hr TL_{50}	29.5	Bond et. al. (1960)
	48-hr TL_{50}	28.5	"
Salmon	48-hr TL_{50}	28.5	Bohmont (1967)
Bluegill	96-hr LC_{50}	35	Gilderhus (1967
	48-hr EC_{50}	19	Cope (1966)
	24-hr TL_{50}	410	Surber and Pickering (196
	48-hr TL_{50}	210	"
	96-hr TL_{50}	140	"
Fathead minnows	24-hr TL_{50}	260	"
	48-hr TL_{50}	220	"
	96-hr TL_{50}	130	"
Striped bass	96-hr TL_{50}	80	Wellborn (1969)
Lake emerald shiner	24-hr TL_{50}	15.7 to >180	Swabey and Schenk (1963
	48-hr TL_{50}	11.7 to 86.2	"
	96-hr TL_{50}	9.1 to 25	"
Rainbow trout	48-hr EC_{50}	20	Cope (1966)
	96-hr LC_{50}	11.2	Gilderhus (1967)
	48-hr TL_{50}	70	Alabaster (1969)
Northern pike	96-hr LC_{50}	16	Gilderhus (1967)
Goldfish	96-hr LC_{50}	35	"
Walleyes	96-hr LC_{50}	2.1	"

TABLE II-32

TOXICITY OF DIQUAT TO AQUATIC INVERTEBRATES

Species	Test	Result (mg/l)	Reference
Amphipod	24-hr TL_{50}	0.58	Wilson and Bond (1969)
	48-hr TL_{50}	0.12	"
	96-hr TL_{50}	0.048	"
Daphnia	24-hr IC_{50}	7.1	Crosby and Tucker (1966)
Mayfly	24-hr TL_{50}	>100	Wilson and Bond (1969)
	48-hr TL_{50}	37	"
	96-hr TL_{50}	16.4	"
Caddisfly	24-hr TL_{50}	>100	"
	48-hr TL_{50}	65	"
	96-hr TL_{50}	33	"
Endipedid	24-hr TL_{50}	>100	"
	48-hr LT_{50}	>100	"
	96-hr TL_{50}	>100	"
Damselfly and Dragonfly	24-hr TL_{50}	>100	"
	48-hr TL_{50}	>100	"
	96-hr TL_{50}	>100	"
Clam	48-hr TL_{50}	>10	Portman and Wilson (1967)
Sand shrimp	48-hr TL_{50}	>10	"

diquat is excreted readily from fish and that residues disappear when the fish are transferred to water containing no diquat.

Toxicity Data Evaluation

Insufficient information is available to assess carcinogenic or teratogenic effects of diquat. Mutagenic data are sufficient to strongly suggest that diquat is not a mutagen. The minority of positive mutagenic results are from less-reliable tests.

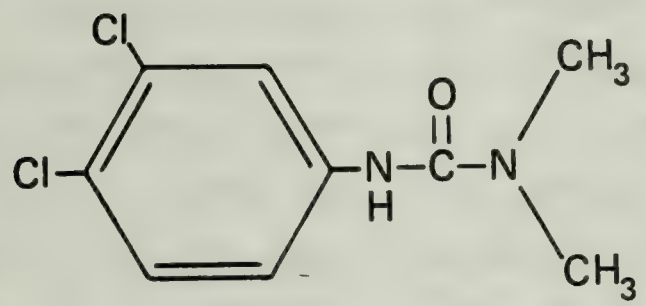
Several studies on diquat were conducted by IBT. A chronic oral test and two studies on reproductive effects have been replaced by Chevron. (Several chronic oral studies and one reproductive study conducted by other laboratories already existed in EPA files.) Regarding two reproduction/residue studies done by IBT, one has been determined to be valid; the other is under EPA review.

EPA is currently identifying data gaps in their registration files.

H. DIURON

1. INTRODUCTION

Diuron is the common name for the herbicide 3-(3,4-dichlorophenyl)-1,1-dimethyl urea (Thomson, 1975) manufactured by Du Pont. It is available commercially in formulations known as dichlorfenidim, DCMV, Di-on®, Diurex-Di-on®, DMU®, Karmex®, Marmex®, and Sup'r-flow (Thomson, 1975), or as Krovar®, a mixture of diuron and bromacil (Ouellette and King, 1977). Diuron has the chemical structure



Diuron is a white solid that is non-corrosive and non-volatile, having a vapor pressure of 0.31×10^{-5} mm Hg at 50°C (Ouellette and King, 1977). Other chemical and physical properties are presented below in the discussion of the fate of diuron in soil and water.

2. TOXICITY

Acute Toxicity

Available information suggests a low order of toxicity for diuron. The LD_{50} for technical diuron is 3400 mg/kg (Hodge et al., 1967). The LD_{50} for Karmex® (80% diuron) is 6964 mg/kg for male rats and 3956 mg/kg for female rats (Du Pont, 1983a). Application of Karmex® to abraded skin of rabbits indicated a dermal LD_{50} of >2000 mg/kg. No irritation or mild irritation occurred when technical diuron was applied to intact or abraded skin of guinea pigs. No irritation was seen when technical diuron (as a 50% aqueous paste) or Karmex® (0.05 g aqueous

paste) was applied to intact human skin (Du Pont, 1983a). A mild hyperkeratosis was reported by Hill et al. (1981) when diuron was applied to rabbit skin at doses of 4.3 mg/ml/day and 3.4 mg/ml/day.

A very mild transient conjunctival irritation was produced by administration of 10 mg of powder or 0.1 ml of a 10% suspension of Karmex® to rabbit eyes (Du Pont, 1983).

Subacute/Subchronic Toxicity

Growth depression and increased erythropoiesis was noted in rats after dietary administration of ten daily doses of 1000 mg/kg diuron (Hodge et al., 1967). No growth depression occurred at 400 ppm in a 90-day feeding study with rats. Growth depression was slight at 2000 ppm and marked at 2500 ppm. In the same study, slight anemia was noted in females at 250 ppm, and at 2500 ppm in both sexes. At 2000 ppm and higher, an abnormal blood pigment was observed (Hodge et al., 1967).

Mammalian Metabolism

No tissue storage occurred in either rats or dogs after they were fed 25-2500 ppm diuron for 9 to 24 months. Excretion in both urine and feces was noted and included N-(3,4-dichlorophenyl) urea as well as small amounts of N-(3,4-dichlorophenyl)-N'-methylurea, 3,4-dichloroaniline, 3,4-dichlorophenol, and unchanged diuron.

Special Studies

Carcinogenicity Limited data suggests that diuron is not carcinogenic. Innes et al. (1969) administered 464 mg/kg diuron by gavage to mice on days 7-28 age, followed by the addition of 1400 ppm in the diet for approximately 18 months. No increase in the incidence of tumors above control values was seen. In a review of available data (including registration material), EPA (1981b) stated that no indication of carcinogenicity was observed in a 2-year feeding study with dogs and a lifetime feeding study with rats.

Teratogenicity/Reproduction Although there is some conflicting evidence, most data indicate that diuron is not teratogenic. When

diuron was administered subcutaneously to mice at a level of 215 mg/kg during days 6 to 14 of gestation, no significant increase was noted in malformations among the offspring of treated mothers (USDHEW, 1969).

In another study, groups of pregnant Wistar rats were given 125, 250, or 500 mg Karmex®/kg on days 6 to 15 of pregnancy via gavage. The Karmex® formulation, which contained 80% diuron, was suspended in corn oil. No signs of maternal toxicity were seen, but maternal body weight was significantly reduced in the 500 mg/kg dams. When rats were killed on day 22, incidences of viable, dead, and resorbed fetuses were comparable to controls. Mean fetal weight, however, was significantly reduced in the top treatment level. An increased frequency in the number of anomalous fetuses was noted at the 250 mg/kg level, but not at 500 mg/kg, when compared to controls. The sole anomaly of statistical significance was an increase in wavy ribs in the top two treatment levels (4.3% and 4.8% for the 250 and 500 mg/kg groups, respectively, compared to 1.5% for controls) (Khera et al., 1979).

No terata were found in two multi-generation rat studies at a dietary concentration of 125 ppm diuron (used in both studies). A weight depression in the second- and third-generation pups was noted in one of the studies (EPA, 1981b; no additional data were available).

Mutagenicity As indicated in Table II-33, diuron does not appear to be a mutagen, although a positive finding with the addition of liver enzymes indicates that further study is needed.

Summary An evaluation of important toxicity data is provided at the end of this chapter.

TABLE II-33

MUTAGENICITY TESTS: DIURON

Test System	Result	Comments	Source
Micronucleus test Mouse bone marrow	-	2000 mg/kg orally	Seiler (1978)
Saccharomyces cerevisiae mitotic gene conversion	-		Fahrig (1974)
Saccharomyces marcescens	-		Fahrig (1974)
Ames/Salmonella	-		Moriya et al. (1983)
	-		Anderson et al. (1972)
	-		Shirasu et al. (1976)
	+	100 µg/plate plus liver in TA 1535	Seiler (1978)
Ames spot test Salmonella typhimurium G46	-	100 µl nitrosated with sodium nitrite in vitro then tested	Seiler (1977)
Escherichia coli WP2	-		Moriya et al. (1983)
	-		Shirasu et al. (1976)
Bacillus subtilis "Rec" assay	-		Shirasu et al. (1976)
rII mutants of T_4 bacteriophage	-	100 µg	Anderson et al. (1972)
Inhibition of testicular DNA synthesis in mouse	+	1000 mg/kg orally	Seiler (1978)
Pelargonium zonale	-	10^{-3}M	Pohlheim and Gunther (1977)

3. MOBILITY AND PERSISTENCE

Fate in Soil and Water

Diuron is considered a low mobility herbicide which remains near the surface of the soil (Harris, 1967; Hill et al., 1955; Hollingsworth, 1955; Miller et al., 1977; Mustafa and Gamar, 1972; Rhodes et al., 1970). In a review of 40 pesticides, Helling (1971a,b,c) found that diuron has low mobility. Majka (1976) determined distribution coefficients to be 14.3 on silty clay loam and 6.5 on loamy fine sand. Both indicate very low mobility. The retention of diuron in soil is suggested by its low solubility (4.2 mg/100 ml water at 25°C--Ouellette and King, 1977), and by the fact that diuron competes successfully with water for adsorption sites in soil, particularly on organic matter (Hance, 1965a).

Lateral and vertical movement of diuron was studied by Ashton (1961) on a peaty much, clay, and sandy loam. In all cases there was limited lateral movement (distances not provided) and vertical movement was restricted to the upper 2.5 cm of soil. Lateral movement was greatest in the sandy loam and least in the peaty muck. In a field study, diuron applied to the surface of a silty clay loam did not move below the 0-5 cm layer after elution with up to 20 cm of water after 54 days (Majka, 1976)

When Reed, (1982) applied 5.66 kg/ha of diuron to a silty clay loam and a sandy loam low in organic matter, diuron remained in the 0-5 layer of soil through out the length of the test (23 days) (amount of water applied not given). Pipe and Cullimore (1980) found that when diuron was added to a heavy clay soil (pH 7.7) at a concentration of 1 ppm, most of the diuron (77%) remained in the top 2 cm, (time not specified).

Other trials, however, have shown higher movement. Ivey and Andrews (1965) showed that phytotoxic concentrations moved 5-10 cm in a Boudre clay loam (3% organic matter) and a Cumberland loam (2.2% organic matter). Increased mobility occurred in soils with less organic matter: phytotoxic concentrations reached 15 cm in a Collins silt loam (1.1% organic matter) and a sequatchie fine sand (1.6% organic matter).

Reed (1982) found movement to 9 cm 8 weeks after application of diuron applied at 5.66 kg/ha. At a higher application rate (13.14 kg/ha) she found movement down to 10-30 cm in a silty loam, a sandy loam, and a sandy loam high in organic matter.

Limited information suggests that diuron may be somewhat persistent in soils. In a study by the Department of Navy (1976) in which diuron was applied at 1-2 kg/ha, the herbicide persisted for 4-8 months. In a study by Ehman and Birdsall (1963), diuron persisted for 5-7 months after application at 2-4 kg/ha (no specification of soil or definition of persistence was provided for either of these studies). After diuron had been applied 32 cotton fields every year for 4-8 years, no phytotoxic residues were found one year after the last application (Dalton et al., 1966).

Diuron is susceptible to microbial breakdown (Dalton et al., 1966; Geissbuhler et al., 1975; Murray et al., 1969). It is possible that microbial breakdown is retarded by adsorption of diuron to soil particles. Geissbuhler et al. (1963) found that desorption of diuron in a humus soil was too slow to maintain a constant rate of degradation by bacteria. Metabolites identified in this study included N-(4-chlorophenoxy)-phenyl-N-methylurea, N-(4-chlorophenoxy)-phenylurea, and (4-chlorophenoxy)-aniline.

In regard to runoff losses, two studies found only trace amounts of diuron in drainage water (Rogers et al., 1974; Willis et al., 1975) (no additional information provided).

Fate in Water

The amount of diuron which will be adsorbed onto aquatic sediments was found to be positively correlated with the amount of organic matter in the sediments (Peck et al., 1980). With high organic matter (for instance, in most wetlands) more diuron is adsorbed, and the diuron is more tightly held. In sediments which are low in organic matter, diuron is readily desorbed. The study also found that there is increased adsorption of diuron in bacterial and fungal cultures were

isolated from pond water and sediments which could degrade diuron. In the laboratory, the mixed cultures could degrade 67%-99% of added diuron. The major metabolite was 3, 4-dichloroaniline. (Ellis and Camper, 1982)

Indicators of Potential Ground Water Contamination

Table II-34 provides information on parameters associated with the mobility of diuron. These parameters, and their associated thresholds, have been suggested by EPA for use in assessing the potential for pesticide contamination of ground water. A discussion of these parameters and thresholds, and the methods for arriving at designated values for individual herbicides, is presented in the main body of the report as part of the discussion of the fate of herbicides in the environment.

TABLE II-34

INDICATORS OF POTENTIAL GROUND WATER CONTAMINATION: DIURON

Indicator	Value for Diuron	Threshold
Solubility	42 ppm at 25°C	>30 ppm
K_{oc}	400	<300-500
Speciation at pH 5	ND*	Anionic (negatively charged)
Hydrolysis half-life	Stable at pH 6-9	>6 months
Photolysis half-life	ND*	>3 days
Vapor pressure	3.1×10^{-6} mm Hg	$<10^{-2}$ mm Hg

*ND = no data

4. TOXICITY TO NON-TARGET ORGANISMS

Birds

Diuron appears to be non-toxic to test birds. Data available are LC_{50} or LD_{50} values from Heath et al. (1972), Hill et al. (1975), and Tucker and Crabtree (1970). The LC_{50} values for mallard ducks, bobwhite quail, Japanese quail, and ring-necked pheasants ranged from 1730 ppm to greater than 5000 ppm, and the LD_{50} value for mallard ducks was greater than 5000 ppm.

Fish

No information was found on the toxicity of diuron to fish.

Lower Aquatic Organisms

Pons and Pussard (1980) reported that 23 strains of amoebae showed no toxic effects when treated with diuron. However, a number of cyanobacteria, green algae, and diatoms were reported to be sensitive to diuron. An abstract by Bednarz and Zarnovski (1980) reported species of Anabaena, Spirulina, Chlorococcum, Chlorella, and Ankistrodesmus to be totally inhibited by 0.1 mg/l. A study by Pipe and Cullimore (1980) showed a population decrease of 99% when the genera Oscillatoria, Chlorella, Stichococcus, Hantzschia, and Navicula were treated with 1 ppm of diuron.

Soil Microorganisms

One study by Chandra et al. (1960) stated that diuron depressed the microbial production of carbon dioxide in a number of different soil types.

Toxicity Data Evaluation

The available carcinogenicity data are insufficient to assess diuron's carcinogenic potential. In their registration standard review, EPA found no studies on the carcinogenicity of diuron which met its requirements; two such tests have been requested by the agency from the manufacturer. Conflicting results in the available data on teratogenic effects do not allow any conclusion to be drawn. Two additional teratogenic tests have been requested by EPA. In regard to mutagenic

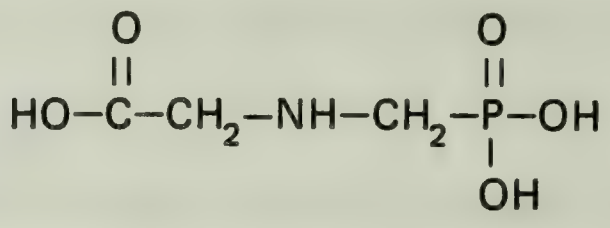
effects, the data in Table II-33 suggest that diuron is not mutagenic, although further tests are needed, as stated above. Three mutagenicity tests are required by EPA's registration standard: a gene mutation study, a chromosomal aberration study, and a study on some other mechanism of mutagenicity. Other toxicity data deficiencies identified by EPA include an acute inhalation test and a dermal sensitization test.

No studies in EPA's registration files on diuron were conducted by IBT.

I. GLYPHOSATE

1. INTRODUCTION

Glyphosate is the common name for the herbicide (N-phosphonomethyl) glycine from Monsanto Chemical Company (Thomson, 1975). It is also called by the trade name Roundup®, a formulation that includes a surfactant (MON 018). Glyphosate has the chemical structure shown below.



Relevant chemical and physical properties are presented below in the discussion of the fate of glyphosate in soil and water.

2. TOXICITY

Acute Toxicity

A low order of toxicity for glyphosate is indicated by acute oral and dermal toxicity studies. Acute oral LD_{50} values for rats, mice, and rabbits are 4320 mg/kg, 4873 mg/kg, and 3800 mg/kg, respectively (Thomson, 1975; Ouellette and King, 1977; and MACC, 1982). Single dermal dosages of 7940 mg/kg did not affect the survival of rabbits (Spurrier, 1973).

Moderate toxicity is indicated by glyphosate when administered intraperitoneally. The LD_{50} values for this route of exposure are 238 mg/kg and 134 mg/kg for rats and mice, respectively (MACC, 1982).

Severe eye irritation is observed in rabbits exposed to Roundup® formulations. In regard to skin irritation, glyphosate itself does not appear to be a skin irritant. However, a mild skin irritation was seen in rabbits after exposure to some formulations of Roundup® (Spurrier, 1973, no data provided).

Mammalian Metabolism

According to a study obtained from EPA registration files (EPA, no publication date provided [d]), glyphosate appears to be readily eliminated. After rabbits were given a single oral dose of ^{14}C-labeled glyphosate, more than 80% of the label was found in feces, and 7-11% was found in urine within 5 days after treatment. Less than 10% was found in expired air. Most of the remaining label was located in the colon.

Special Studies

Monsanto (1982) reports that no signs of carcinogenicity, teratogenicity, or neuropathology and no adverse reproductive effects from glyphosate were seen in a 2-year rat study, an 18-month mouse study, and a 2-year dog study.

A number of studies have been published on the mutagenic potential of glyphosate. As summarized in Table II-35, most of these studies indicate that glyphosate is not a mutagen. Two studies show questionable positive responses. One of these, by Vigfusson and Vyse (1980), showed some sister chromatid exchange upon exposure of human lymphocytes to relatively high concentration of 0.25 mg/ml, 2.5 mg/ml and 25 mg/ml glyphosate. No clear dose response was evident. Furthermore, only two donors were used for lymphocyte samples. Lymphocyte sister chromatid exchange is known to vary considerably between subjects. The other positive result came from an Ames/Salmonella spot test in which exposure to nitrosated glyphosate resulted in a slight increase in mutations at 1 µl and 10 µl, but not at 100 µl (Seiler, 1977). The author does not state whether the lack of response at 100 µl is due to toxicity or to a poor dose response.

Summary An evaluation of important toxicity data is provided at the end of this chapter.

TABLE II-35

MUTAGENICITY TESTS: GLYPHOSATE

Test System	Result	Comments	Source
Sister chromatid exchange in human lymphocytes *in vitro*	(+)*	See text	Vigfusson and Vyse (1980)
Drosophila sex-linked recessive lethal	-		Gopalan and Njagi (1981, abstract)
Ames/*Salmonella*	-		Moriya et al. (1983)
	-	25 µg/plate plus liver homogenate	Wildeman and Nazai (1982)
	-	25 µg/plate plus plant homogenate	"
Ames spot test *Salmonella typhimurium* G46	(+)	See text; glyphosate nitrosated with sodium nitrite *in vitro*	Seiler (1977)
Esherichia coli WP2	-		Moriya et al. (1983)

* Parentheses indicate uncertainty.

3. MOBILITY AND PERSISTENCE

Fate in Soil

Glyphosate binds tightly to soil particles (TRW, 1981). Helling (1971a,b,c) states that glyphosate is readily adsorbed to all soils, and that it is classified as immobile by the Helling and Turner classification system. Adsorption of glyphosate begins immediately on contact with the soil, and binding is rapid onto clays (kaolinite, illite, and bentonite) and muck. Monsanto performed soil column leaching studies in which soil columns were treated with glyphosate or its sodium salt and then aged for 30 days (EPA, no publication date provided [d]). Upon elution with 0.5 acre-inch of water for 45 days, the leaching of glyphosate was said to be insignificant.

Adsorption of glyphosate is affected by a number of factors. Phosphates compete with glyphosate for binding sites, so adsorption is greater in low-phosphate soils (Sprankle et al., 1975a). Adsorption is greater in Fe^{+++} and Al^{+++} saturated soils than in Na^{+} or Ca^{++} saturated soils. As would be expected, glyphosate was found to be less tightly bound to sand than to other soils (Sprankle et al., 1975).

A review by TRW (1981) concludes that glyphosate dissipates rapidly in soil. In a study of microbial degradation, Reuppel et al. (1977) found that glyphosate was degraded almost as rapidly as sucrose. When both were labeled, 47% to 55% of the glyphosate radioactivity was given off as CO_2 in 4 weeks, compared to 57.9% for sucrose. This study found the major soil metabolite to be aminomethyl phosphonic acid (AMPA). Several other metabolites were also detected, all at less than 1% of the original glyphosate concentration. These degradation products include N-methylaminomethyl phosphonic acid, glycine, N,N-dimethylaminomethyl phosphonic acid, and hydroxymethyl phosphonic acid. Rueppel et al. (1977) state that based upon shake tests, AMPA is highly biodegradable, although the rate may be slower than that of glyphosate, possibly due to tighter binding to the soil and/or lower permeability through the microbial cell walls.

Other information suggests a more variable persistence. Reuppel et al. (1977) studied the dissipation of glyphosate on a silty clay loam, a silt loam, and a sandy loam. When glyphosate was applied to the three soils at rates of 4 ppm and 8 ppm, the half-lives on the three soils were found to be 3, 27, and 130 days, respectively, independent of application rate. Studies by Monsanto and others (EPA, no publication date provided [d]) report half-lives in a variety of soils ranging from 8 to 19 weeks.

Reuppel et al. (1977) performed runoff potential experiments using a silty loam, a silty clay loam, and a sandy loam in soil beds inclined at an angle of 7.5°. Glyphosate was applied to the upper third of the soil surface at a rate of 1.12 kg/ha. When artificial rainfall was applied (amounts not given) at 1-, 3-, and 7-day intervals, it was shown that the maximum runoff that would occur would be less than 0.02% of the original herbicide applied.

Persistence in Water

There have been very few studies concerning the persistence of glyphosate in water. The data suggest that glyphosate is slowly degraded in aqueous systems. In a study by Brightwed and Malik (no date provided), 0.1 ppm glyphosate was added to water samples from a sphagnum bog (pH 4.23), a cattail swamp (pH 6.25), and pond water (pH 7.33). After incubation in the dark for 49 days, the half-lives of glyphosate were calculated to be 7, 9, and 10 weeks, respectively. Serdy (1980) states that glyphosate is adsorbed to mineral and organic matter and degraded by microorganisms.

Indicators of Potential Ground Water Contamination

Table II-36 provides information on parameters associated with the mobility of glyphosate. These parameters, and their associated thresholds, have been suggested by EPA for use in assessing the potential for pesticide contamination of ground water. A discussion of these parameters and thresholds, and the methods for arriving at designated values for individual herbicides, is presented in the main

body of the report as part of the discussion of the fate of herbicides in the environment.

TABLE II-36

INDICATORS OF POTENTIAL GROUND WATER CONTAMINATION: GLYPHOSATE

Indicator	Value for Glyphosate	Threshold
Solubility	12,000 ppm at 25°C	>30 ppm
K_{oc}	ND*	<300-500
Speciation at pH 5	Anionic	Anionic (negatively charged)
Hydrolysis half-life	ND*	>6 months
Photolysis half-life	ND*	>3 days
Vapor pressure	Negligible	$<10^{-2}$ mm Hg

*ND = no data.

4. TOXICITY TO NON-TARGET ORGANISMS

Birds

One feeding study by Serdy (1980) showed glyphosate to be non-toxic to mallard ducks and quail, with LC_{50} values of greater than 4600 ppm.

Fish

A study by Folmar et al. (1977) found that the 96-hr LC_{50} values for Roundup® ranged from 2.3 mg/l for fathead minnows to 13 mg/l for channel catfish (see Table II-37). The data presented in this table suggest that it is the surfactant (MON 0818), not the glyphosate, that is the primary toxic agent in Roundup®.

TABLE II-37

TOXICITY OF GLYPHOSATE TO FISH

Species	Formulation	Water Temp. (°C)	TL_{50} or LC_{50} (mg/l) and 95% Confidence Limits 24-hr	96-hr	Source
Rainbow trout	Roundup®	7	14 (11-17)	14 (11-16)	Folmar et al. (1977)
		17	7.5 (6.3-9.0)	7.4 (6.2-8.9)	
	Glyphosate	12	140 (120-170)	140 (120-170)	"
	Surfactant	12	2.1 (1.6-2.7)	2.0 (1.5-2.7)	"
Fathead minnow	Roundup®	22	2.4 (2.0-2.9)	2.3 (1.9-2.8)	"
	Roundup®	Not given		9.4	Monsanto (no date [a])
	Glyphosate	22	97 (79-120)	97 (79-120)	Folmar et al. (1977)
	Surfactant	22	1.4 (1.2-1.7)	1.0 (1.2-1.7)	"
Channel catfish	Roundup®	22	13 (11-16)	13 (11-16)	"
	Glyphosate	22	130 (110-160)	130 (110-160)	"
	Surfactant	22	18 (8.5-38)	13 (10-17)	"

TABLE II-37 (CONTINUED)

TOXICITY OF GLYPHOSATE TO FISH

Species	Formulation	Water Temp. (°C)	TL_{50} or LC_{50} (mg/l) and 95% Confidence Limits		Source
			24-hr	96-hr	
Bluegill	Roundup®	17	9.6 (7.9-12.0)	7.5 (6.3-9.0)	Folmar et al. (1977)
		22	6.4 (4.8-8.6)	5.0 (3.8-6.6)	"
		27	4.3 (3.4-5.4)	4.0 (3.2-5.0)	"
	Glyphosate	22	150 (120-190)	140 (110-160)	"
	Surfactant	22	3.0 (2.5-3.7)	3.0 (2.5-3.7)	"
Carp	Roundup®	Not given		3.9	Monsanto (no date [a])
Catfish	Roundup®	Not given		16	"
Trout	Roundup®	Not given		11	"

Folmar et al. (1977) also exposed rainbow trout and channel catfish to Roundup® at various stages of development. For both species, the egg stage was least sensitive to Roundup®, and sensitivity increased in the sac fry and early swim-up stages, then decreased as the fishes aged. Trout eggs exposed to 10 mg/l showed a significant reduction in the percentage of eggs that hatched. No significant difference was noted at 5 mg/l. A significant number of sac-fry were killed at 5 mg/l, but not at 2 mg/l. The author concluded that applications of Roundup® could have adverse effects if applied when young fish were present.

In the material safety data published by Monsanto (no publication date provided [a]), Roundup® was referred to as being slightly or moderately toxic to bluegill, carp, catfish, fathead minnows, and trout. The LC_{50} or TL_{50} values ranged from 3.9 mg/l for carp to 16 mg/l for catfish. Monsanto also reports that carp were unaffected for a period of 90 days following exposure to an aerial application of Roundup® at the intended use level in a static pond.

Higher temperatures increase the toxicity of glyphosate to fish, as shown in Table II-38. Roundup® is about twice as toxic to rainbow trout at 17°C as at 7°, and it is more toxic to bluegills at 27° than at 17° (Folmar et al. 1977). The effect of pH is less clear. Increasing pH results in a decrease in the toxicity of glyphosate alone. Increasing pH, however, results in a decrease in toxicity of Roundup® or the surfactant alone.

Lower Aquatic Organisms

As summarized in Table II-39, the effects of glyphosate and its formulations were investigated by Folmar et al. (1977). The 48-hr LC_{50} or EC_{50} values for Roundup® range from 3.0 mg/l for daphnids to 60 mg/l for scud. The data indicate that the surfactant was more toxic than the glyphosate in Roundup®.

In the material safety data published by Monsanto (publication date not provided [a]), Roundup® was referred to as moderately toxic to

TABLE II-38

EFFECTS OF TEMPERATURE ON TOXICITY OF GLYPHOSATE TO FISH

Species	Water Temp. (°C)	TL_{50} or LC_{50} (mg/l) and 95% confidence limits		Source
		24-hr	96-hr	
Rainbow trout	7	14(11-17)	14(11-16)	Folmar et al. (1977)
	12	14(11-17)	7.5(6.3-9.0)	"
	17	7.5(6.3-9.0)	7.4(6.2-8.9)	"
Bluegill	17	9.6(7.9-12.0)	7.5(6.3-9.0)	"
	22	6.4(4.8-8.6)	5.0(3.8-6.6)	"
	27	4.3(3.4-5.4)	4.0(3.2-5.0)	"

TABLE II-39

TOXICITY OF GLYPHOSATE TO LOWER AQUATIC ORGANISMS

Species	Formulation	Water Temp. (°C)	TL_{50} or LC_{50} (mg/l) and 95% confidence limits 24-hr	48-hr	96-hr	Source
Daphnids	Roundup®	22		3.0(2.6-3.4)		Folmar et al. (1977)
Scud	Roundup®	12	100	62(40-98)	43(28-66)	"
Midge larvae	Roundup®	22		18(9.4-32)[1]		"
	Glyphosate	22		55(31-97)[1]		"
	Surfactant	22		13(7.1-24)[1]		"
Crayfish	Roundup®	Not given			>1000	Monsanto (no date [a])
Daphnia	Roundup®	Not given		5.3		"
						"

[1] Midge toxicity expressed as 48-hr EC_{50}.

Daphnia (the 48-hr LC_{50} was 5.3 mg/l) and practically non-toxic to crayfish (the 96-hr LC_{50} was >1000 mg/l).

Bees

Glyphosate appears to be non-toxic to bees. An experiment with bees by Serdy (personal communication) indicated an LD_{50} of greater than 100 µg/bee. Spurrier (1973) reported that honeybees could tolerate up to 100 µg/bee for 48 hours (topically or orally). No study was cited. The same data were reported in material safety data published by Monsanto (date of publication not given).

Soil Microorganisms

A report by Reuppel et al. (1977) concluded that glyphosate appeared to have no effect on the total microflora population. Quilty and Geoghegan (1976) found glyphosate to have a minimal effect on microflora in peat. A review of registration files by the EPA (no publication date provided [d]) concluded that glyphosate showed no apparent effect on nitrification or nitrogen fixation by microbes, or on degradation of starch, cellulose, protein, or leaf litter.

Bioaccumulation

In the material safety data published by Monsanto (no publication date provided [d]), tissue residue analyses indicated that glyphosate does not bioaccumulate in carp exposed for 90 days to an intended use level of Roundup® aerially sprayed on a static pond. Similar conclusions were drawn by Monsanto (no publication date provided [b])). A study by Folmar et al. (1977) reported the effects of exposing rainbow trout to 0.02, 0.2, and 2.0 mg/l to the isopropylamine salt of glyphosate or Roundup® for 12 hours. No residues were found at the two lower concentration, but at at the highest level, (2.0 mg/l), fillets contained 80 µg/kg and the eggs contained 60 µg/l of glyphosate.

Monsanto (1982) states that glyphosate's high water solubility and low lipid solubility suggests that it should not bioaccumulate and that it should not accumulate in the event of repetitive exposure.

Toxicity Data Evaluation

Most of the concern about data regarding glyphosate toxicity has focused on the tests done by IBT. The following studies done by IBT on glyphosate were determined to be invalid and have been replaced by Monsanto:

dermal (rabbit) subchronic (3 studies)
chronic oral (rat)
carcinogenicity (mouse)
mutagenicity (mouse)
teratology (rabbit)
mutagenicity (Ames test)
recombination assay
teratology (rabbit)
subchronic inhalation (rat)
dermal (rabbit)

The following studies were found to be valid:

reproduction (rat)
subchronic oral (rat)
subchronic oral (dog)
chronic oral (dog)
mutagenicity (rat/mouse)
reproduction/residue (hen)
dermal (quail)
dermal (swine)
dermal (cattle)
dermal (hen) (2 studies)

EPA has decided that a subchronic oral (rabbit) and a cholinesterase (rat) study are no longer required. The agency is still reviewing a pilot and chronic feeding study (rat) and a chronic oral (dog) study.

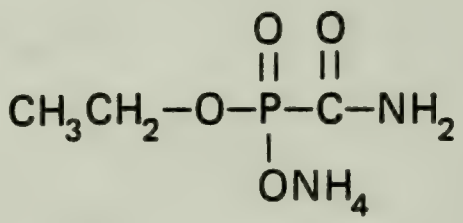
Monsanto has given EPA a negative response in regard to its intention to replace a mutagenicity study (mouse) and a teratology study (rabbit). The company has not yet responded in regard to its intention to replace a reproduction (rat) study.

More publicly available information is needed to allow an independent review of glyphosate. Because glyphosate was registered since the 1972 data requirements, however, it can be assumed that, once the IBT data replacement is completed, the full complement of data will have been reviewed and found acceptable by EPA.

J. KRENITE

1. INTRODUCTION

Krenite® is the trade name for the herbicide ammonium ethyl carbamoyl phosphonate, produced by E. I. du Pont de Nemours and Company (Thomson, 1975). Its common name is fosamine ammonium, and its structure is



Krenite® contains 41.5% fosamine ammonium (4 lb/gallon) (Du Pont, 1983). Relevant physical and chemical characteristics are presented below in the discussion of the fate of Krenite® in soil and water.

2. TOXICITY

Acute Toxicity

Krenite® appears to have low acute toxicity. Toxicological information supplied to Du Pont by Sherman (1979) cited oral LD_{50} values for the 42% aqueous solution of the active ingredient, fosamine ammonium, to be 24,000 mg/kg for male rats, 7,380 mg/kg for guinea pigs, and >15,000 mg/kg for female beagle dogs. Sherman (1979) studied dermal toxicity and reported that a maximum feasible dose of 1680 mg/kg resulted in no signs of toxicity in New Zealand rabbits. The USDA (1978) reported an acute dermal LC_{50} value for fosamine ammonium to be >4000 mg/kg for rabbits.

In regard to inhalation toxicity, Sherman (1979) found that fosamine ammonium concentrations of 56.6 mg/l and 42.0 mg/l showed no signifi-

cant clinical signs of toxicity to female rats exposed to 1 hour of the 42% aqueous solution in aerosol form. In a test for eye and skin irritation, New Zealand rabbits exposed to 10 ml of the 42% aqueous solution showed mild to moderate erythema after a 24-hour exposure, but after 72 hours, appearance was normal. New Zealand rabbits showed no ocular effects when exposed to 0.1 ml of the 42% aqueous solution of fosamine ammonium 24, 48, and 72 hours after treatment (Sherman, 1979).

Fosamine ammonium formulations often contain non-ionic surfactants such as Tween 20®, Triton X-100®, or Du Pont Surfactant WK®. OSHA data sheets and information supplied by the manufacturers state that these materials are not considered hazardous or toxic (TRW, 1981).

Special Studies

Carcinogenicity No data are available.

Teratogenicity/Reproduction Sherman (1979, unpublished; no data provided) fed 0, 200, 1000, and 10,000 ppm fosamine ammonium to Charles River-CD rats on days 6 through 15 of gestation. No signs of embryotoxicity or teratogenicity were observed, based on the absence of internal, skeletal, or external abnormalities or malformations.

Mutagenicity No indications of mutagenic potential were seen in an E. coli WP2 test system and an Ames/Salmonella test system using strains TA-98 and TA-100 (Moriya et al., 1983). Sherman (1979, unpublished; no data provided) found no evidence of mutagenicity in an Ames/Salmonella test using strains TA-1535 and TA-100 to detect base-pair substitution mutations and strains TA-1537, TA-1538, and TA-98 to detect frame shift mutations.

Summary An evaluation of important toxicity data is provided at the end of this chapter.

3. MOBILITY AND PERSISTENCE

Fate in Soil

Fosamine ammonium is considered to have low mobility in soil, despite its high water solubility (179 g/100g; no temperature given) (TRW,

1981). This is due to a strong tendency to adsorb to soil particles. Fosamine ammonium was found to have a Freundlich equilibrium constant of greater than 20 on Keyport silt loam (17% clay, 2.8% organic matter) (Du Pont, 1975. A standard textbook on ground water movement (Freeze and Cherry, 1979) states that a Freundlich equilibrium constant of greater than 1 indicates that a substance is essentially immobile in porous media (such as soil). EPA considers values greater than 1 to 5 as indicative of a low potential for ground water contamination (Servern, 1983).

The potential for movement of fosamine ammonium was studied under simulated rainfall conditions, using a sandy loam (12% clay) in a flat (12" × 36" × 3") sloped at 5°-10°. Water was applied for 2 hours at a rate of 12.5-25 ml/hr. The top third of the flat was treated with 15 lb/acre fosamine ammonium. Most (92.6%) remained in the first inch of soil. TRW (1981) states that several other field studies (EPA no publication date provided [b]; Han, 1979b; Mullison, 1979), for which no data are presented, also confirm that fosamine ammonium has a low vertical mobility. Soils with higher adsorption capacities will tend to retard fosamine ammonium movement more than soil with lower adsorption capacities. In a laboratory leaching test using silt loam (17% clay) and a sandy loam (12% clay), fosamine ammonium moved more in the soil with the lower clay content, although mobility was low in both soils (TRW, 1981; no primary source given).

Because fosamine ammonium tends to stay near the soil surface, erosion or runoff may lead to lateral movement of the herbicide, especially after a heavy rainfall (TRW, 1981; no primary source given).

Fosamine ammonium is not considered to be persistent in soils. It has a half-life of approximately one week (Han, 1979b) to 10 days (Mullison 1979). Han (1979b) documented that 11.3 kg/ha of fosamine ammonium applied to a silt loam (17% clay, 2.8% organic matter) had a half-life of approximately 1 week. In a heavier silt loam (31% clay, 4% organic matter) it had a half-life of less than 1 week. In two silt loams and a fine sand, fosamine ammonium and its major metabolite carbamoyl-

phosphonic acid (CPA) were not detected after 3 to 6 months (Han, 1979).

Theoretically, fosamine ammonium can be degraded to CPA by chemical hydrolysis (Han, 1979b; Mullison, 1979). However, Han (1979b) showed that under sterile soil conditions fosamine ammonium was not degraded in the first 20-30 days and only minimally thereafter. Under unsterile conditions, degradation was fairly rapid (20%-25% of the original weight of ^{14}C was evolved in the first 20-30 days). Thus, it appears that the degradation of fosamine ammonium in the soil is predominantly due to microbial action.

When degradation rates were compared in two silt loams and a fine sand, it was found that fosamine ammonium was metabolized to CPA more quickly in the fine sand (Leon Immokalee fine sand, with 99% sand and 1% organic matter), although the subsequent degradation of CPA was somewhat slower in the fine sand than in the silt loams (Han, 1979b).

Persistence in Water

The degradation of fosamine ammonium in water appears to be strongly pH-dependent (Han, 1979b). At a pH of 5, fosamine ammonium at 5 ppm was hydrolyzed nearly completely to CPA within 2 weeks, with a half-life of approximately 10 days. At pH 7 and 9, the same concentration of fosamine ammonium was found to be stable for 4 weeks (less than 3% decomposition). These laboratory studies were conducted in the dark, using labeled fosamine ammonium. The author concludes that decomposition will be minimal under field conditions; however, the slightly acidic nature of many Massachusetts waters may increase decomposition.

Photolysis of fosamine ammonium appears to be minimal under both field and laboratory conditions. At an aqueous concentration of 5 ppm at pH 5, photodegradation was "very minor" after 4 weeks in direct July sunlight in Wilmington, Delaware (Han, 1979b). In a laboratory study, when an aqueous solution of 5 ppm fosamine ammonium was irradiated at

an intensity of 1200 watts/sq cm, only 2% decomposition occurred after 8 weeks (Han, 1979b).

Indicators of Potential Ground Water Contamination

Table II-40 provides information on parameters associated with the mobility of fosamine ammonium. These parameters, and their associated thresholds, have been suggested by EPA for use in assessing the potential for pesticide contamination of ground water. A discussion of these parameters and thresholds, and the methods for arriving at designated values for individual herbicides, is presented in the main body of the report as part of the discussion of the fate of herbicides in the environment.

TABLE II-40

INDICATORS OF POTENTIAL GROUND WATER CONTAMINATION: FOSAMINE AMMONIUM

Indicator	Value for Fosamine Ammonium	Threshold
Solubility	1,790,000 ppm at 25°C	>30 ppm
K_{oc}	ND*	<300-500
Speciation at pH 5	ND*	Anionic (negatively charged)
Hydrolysis half-life	2 weeks**	>6 months
Photolysis half-life	ND*	>3 days
Vapor pressure	4×10^{-6} mm Hg at 25°C	$<10^{-2}$ mm Hg

* ND = no data
** To CPA at 24° and pH 5.5-6.5.

4. TOXICITY TO NON-TARGET ORGANISMS

Birds

Studies by Mullison (1979) and E. I. Du Pont de Nemours Company (1979) using mallard ducks and bobwhite quail showed fosamine ammonium to have low toxicity for these species. The LD_{50} values for both species were greater than 10,000 mg/kg.

A study by Sherman (1979) reported the acute LD_{50} values for mallard ducks and bobwhite quail to be greater than 5000 mg/kg for both species of birds. The same report cited LC_{50} subacute toxicities for both species to be greater than 10,000 ppm.

Fish

Fosamine ammonium appears to be non-toxic to fish. A study by the U.S. Department of the Interior (1978) showed fosamine ammonium to have a low toxicity for bluegills, rainbow trout, and fathead minnows, with LC_{50} values ranging from 670 ppm to greater than 1000 ppm. A static bioassay by Du Pont (1980) on salmon indicated a 96-hour LC_{50} of 8290 ppm.

Lower Aquatic Organisms

One study by Du Pont (1980) shows fosamine ammonium to be non-toxic to Daphnia, with a 48-hr LC_{50} of 1524 ppm.

Bees

Fosamine ammonium appears to be non-toxic to bees. A solution with 10,000 ppm produced no greater mortality than that seen in a control population of bees, according to a study by Du Pont (1980).

Soil Microorganisms

Non-photosynthetic microorganisms seem to be relatively unaffected by fosamine ammonium. EPA (publication date not provided [b]) state that in three types of soils, populations of various bacteria and fungi remained unaltered over a 8-week period after treatment with 10 ppm fosamine ammonium. The same report showed little or no fungal toxicity at rates up to 100 ppm when using various species of fungi (Aspergillus

niger, A. terreus, Penicillium citrinum, Gibberella aubinetti, Fusarium sp., Alternaria sp., Rhizoctonia solani, and Pythium sp.). Soil-nitrifying bacteria in two different soils remained unaffected during a 5-week period after treatment with 0.5, 5, and 20 ppm fosamine ammonium in studies by Han (1979) and by Han and Krause (1979). However, Hallborn and Bergman (1979) reported that the rate of nitrogen fixation by the lichen Peltigera praetextata and its free-living phycobiant algae, Nostoc sp., was drastically reduced, with total inhibition occurring after 8 hours.

Potential for Bioaccumulation

Moore (1976) reports that fosamine ammonium is considered to have a low potential for bioaccumulation. Han (1979a) showed that fosamine ammonium and its soil degradation product, carbamoylphosphonic acid, did not accumulate in channel catfish when they were exposed to 1 ppm fosamine ammonium for 4 weeks. The accumulation factor was less than 1, and 50% of these residues were eliminated after 2 weeks.

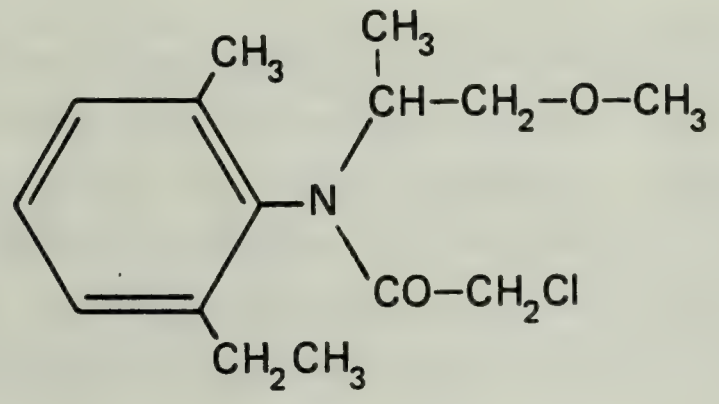
Toxicity Data Evaluation

More publicly available information is needed on fosamine ammonium. However, since it was registered after the 1972 data requirements were in place, it can be assumed that the manufacturer conducted the complement of tests required by EPA at that time and that the results were found to be acceptable by EPA. No tests were conducted by IBT.

K. METOLACHLOR

1. INTRODUCTION

Metolachlor is the common name for the herbicide 2-chloro-N- (2-ethyl-6-methylphenyl)-N-(2-methoxy-1-methylethyl) acetamide. (American National Standards Institute, 1976), produced by Ciba-Geigy Chemical Company. It is also called Dual®, Ontrack®, and the experimental number CGA-24705. It has the chemical structure



Relevant chemical and physical properties are presented below in the discussion of the fate of metolachlor in soil and water.

2. TOXICITY

Acute Toxicity

Metolachlor exhibits a low order of toxicity in acute tests. Bathe (1973) reports the LD_{50} value for technical metolachlor for rats to be 2780 mg/kg. Several studies have been done of the 6 lb and 8 lb per gallon emulsifiable concentrate (EC) formulations. The acute oral LD_{50} value for the 6 lb/gallon EC formulation was found to be between 2000 and 5000 mg/kg for rats by Affiliated Medical Research, Inc. (1974a). Nham and Harrison (1977a) reported the LD_{50} for the 8 lb/gallon EC to be 2530 mg/kg.

Low acute dermal toxicity was indicated in a study by Affiliated Medical Research, Inc. (1974b) which found an LD_{50} of greater than 10,000

mg/kg when technical metolachlor was applied to unabraded rabbit skin. The same study found an acute dermal LD_{50} of one 6 lb/gallon EC formulation to be also greater than 10,000 mg/kg. Nham and Harrison (1977b) established the LD_{50} for rabbits to be greater than 3038 mg/kg via the intact dermal route.

Low inhalation toxicity was found by Sachsse and Ullman (1974), who observed no deaths of rats after a 4-hour exposure to 1.752 mg/l (maximum achievable level) of the technical form.

Sachsse (1973a) found metoloachlor to be non-irritating (irritation index of 0.1) when technical metolachlor was applied to the skin of rabbits. Mild irritation (irritation index of 1.62) was observed for one 6 lb/gallon EC formulation by Affiliated Medical Research, Inc. (1974c). Scribor (1977b) reported moderate erythema, edema, and second-degree burns after a 72-hour treatment with the 8 lb/gallon EC formulation.

Technical metolachlor (0.1 ml) was found to be non-irritating to rabbit eyes after 24 hours and after 7 days in a study by Sachsse (1973b).

Subchronic Toxicity

In their registration standard review of metolachlor, EPA (1980) reported problems with a number of subchronic studies. One study considered valid was performed on dogs for 6 months and showed that metolachlor produced no observable effects at a dietary dose of 100 ppm. When 540 mg/kg/day of metolachlor 6E (68.5% a.i.) was applied to the skin, no significant evidence of systemic effects was noted. At 1080 mg/kg/day, the only reported effect was decreased body weight gain (Affiliated Medical Research, Inc., 1974d).

Special Studies

Carcinogenicity In its registration standards review of metolachlor, EPA (1980) cited two studies which showed no evidence that metolachlor is a carcinogen, although further testing is needed. One study cited by EPA (Gesme et al., 1977) was conducted by Industrial Bio-Test Laboratories (IBT), and validated later by Ciba-Geigy and EPA after an

in-depth evaluation. The study showed no evidence of carcinogenicity after feeding 50 male and 50 female Charles River CD-1 rats at dietary dosages of 30, 1000, and 3000 ppm metolachor. Although several deficiencies in animal husbandry and good laboratory practices were noted, EPA decided that the negative results were supported by the raw data.

The other study cited by EPA (1980) is a 2-year feeding study on the rat which also reported no evidence that metolachlor is carcinogenic (Kennedy, 1976). These results, however, are considered only "supplementary" by EPA, because of significant deficiencies in test protocol, including a failure to verify the dose levels by an analysis of the diet.

Teratogenicity/Reproduction No fetotoxic effects or effects on offspring were noted after 60, 180, and 360 mg/kg/day of metolachlor were administered to female Sprague-Dawley rats during days 6 to 15 of gestation. The only effect noted was a decrease in food consumption at the highest dose in the early part of the experiment. A study by Smith and Adler (1978) found no reproductive effects of metolachlor on the rat. EPA considered the conclusions of the test to be only "supplementary" information, because of several deficiencies in the test, including problems in animal husbandry, mating performance and success, and observation records.

Mutagenicity

Two tests cited by EPA (1980) showed no evidence of mutagenic activity of metolachlor. Arni and Miller (1976) tested metolachlor in a bacterial (Salmonella) system, utilizing activation by mammalian microsomes. No increase in base substitutions or point mutations was observed in comparison to controls at a range of 10, 100, 1000, and 10,000 µg/plate. The effect of metolachlor on developing sperm was investigated by Ciba-Geigy Limited (1976) in an in vivo mouse study using single oral doses of 100 and 300 mg/kg metolachlor. No effect was observed on fertility rates or on zygote or embryo survival. No malformations of resulting embryos were noted.

Neurotoxicity Since metolachlor is a chloracetanilide herbicide, it is not expected to cause esterase depression or delayed neurotoxicity (EPA, 1980). No test for neurotoxicity is required for metolachlor by EPA.

Summary An evaluation of important toxicity data is provided at the end of this chapter.

3. MOBILITY AND PERSISTENCE

Fate in Soil

The information discussed below is from the EPA pesticide registration standard for metolachlor issued in 1980, which reviews information submitted by the manufacturer as well as information which is publicly available.

Metolachlor appears to have a significant potential for movement in soil. Its water solubility is high (530 ppm at 20°C). In a column study done by Houseworth (1973), "extensive leaching" of metolachlor was observed in soils having a low percentage of organic matter. Dupre (1974a) and Houseworth (1973) document that leaching will readily occur in sandy loam and sandy soils that are low in organic matter (<2%). In the latter study, 20% to 33% of the applied metolachlor leached more than 30 cm (12 inches) in the soils when an equivalent of 20 inches of rainfall was applied to a column that was overlain by metolachlor. Ballantine (1975) showed substantial leaching of metolachlor and its metabolites (by anaerobic degradation) into the 6-inch to 12-inch soil horizon in five soil types. In a study by Skipper, Gossett, and Smith (1976), extensive leaching was considered to be the major cause of disappearance of metolachlor from the upper 3 inches of a sandy loam soil (no data provided).

In field dissipation studies (Ballantine, 1975) 2 and 4 lb a.i./acre of metolachlor were added to five soils: a "Mississippi loam," a "Nebraska silt loam," and three unnamed soils from New York, California, and Illinois. In the top 12 inches of soil metolachlor dissipated to 10% of the original dose after 60 to 162 days. Part of this loss would be due to microbial degradation, both aerobic and anaerobic (McGahen and Tiedje, 1978). In a clay loam, Elleghausen (1976a, b) found 90% degradation under non-sterile conditions.

Degradation products may also be mobile. Dupre (1974b) reports that "residues of aged 14-C-Metolachlor" were observed to leach in sandy loam soil. The results suggested that several different chemicals were involved, each with a different mobility.

Dupre (1974a) conducted a runoff study which indicated that both sheet erosion and leaching are probably involved in the movement of metolachlor (no supporting data provided).

The hydrolysis half-life of metolachlor is 200 days over a pH range of 5 to 9, indicating considerable stability in regard to this route of degradation. Photolysis may be more important, given a 50% degradation in sunlit soil after 8 days (Aziz, 1974). However, EPA (1980) considers photolysis to be an insignificant route of loss if metolachlor is incorporated into the top 2 inches of soil. Volatility is probably not a significant route of loss, since metolachlor has a vapor pressure of 1.3×10^{-5} mm Hg at 20°C.

At the end of the its discussion of the fate of metolachlor in soil, EPA (1980) states that "This high mobility, in combination with a potential for long-term environmental stability, may prove to be [a] significant concern in projecting potential exposures to Metolachlor residues."

Fate in Water

The only data available on the fate of metolachlor in water concerns its degradation by hydrolysis and photolysis, both of which are insignificant. In buffered solutions at a temperature of 30°C and at pH levels of 5,7, and 9, metolachlor was stable for 28 days (Burkhard, 1974). The percentages remaining at each pH level were 97, 100, and 96, respectively.

Aziz and Kahrs (1975) found metolachlor to be relatively stable in aqueous solutions exposed to natural sunlight. After 30 days, only 6.6% of the original concentration had been photolyzed.

Indicators of Potential Ground Water Contamination

Table II-41 provides information on parameters associated with the mobility of metolachlor. These parameters, and their associated thresholds, have been suggested by EPA for use in assessing the potential for pesticide contamination of ground water. A discussion of these parameters and thresholds, and the methods for arriving at designated values for individual herbicides, is presented in the main body of the report as part of the discussion of the fate of herbicides in the environment.

TABLE II-41

INDICATORS OF POTENTIAL GROUND WATER CONTAMINATION: METOLACHLOR

Indicator	Value of Metolachlor	Threshold
Solubility	530 ppm at 20°C	>30 ppm
K_{oc}	178.4	<300-500
Speciation at pH 5	Cationic* (positively charged)	Anionic (negatively charged)
Hydrolysis half-life	>200 days**	>6 months
Photolysis half-life	Slow (8% after 30 days)	>3 days
Vapor pressure	1.3×10^{-5} mm Hg at 20°C	$<10^{-2}$ mm Hg

* By analogy with trimethylamine.
** At 20°C and pH 5-9.

4. TOXICITY TO NON-TARGET ORGANISMS

Birds

Two studies by Fink assessing the effect of metolachlor on birds are reported by EPA (1980) as part of their generic standards review. Fink (1974a, b) reported dietary LD_{50} values for mallard ducks and bobwhite quail to be greater than 10,000 ppm, indicating that

metolachlor was practically non-toxic to upland game birds and waterfowl.

Fink (1978a, b) also studied the effect of metolachlor administered for 1 week to quail and mallard ducks. At most dosages, significantly fewer chicks survived to 14 days, as shown in Table II-42.

Fish

EPA (1980) concluded that metolachlor was moderately toxic to fish. One study by Buccafusco (1978a, b) reported 96-hr LC_{50} values for bluegills and rainbow trout to be 10.0 ppm and 3.9 ppm, respectively.

Dionne (1978) found the no effect level, below which no effects were observed, to be between 0.78 and 1.60 ppm.

TABLE II-42

EFFECT OF METOLACHLOR ON REPRODUCTIVE SUCCESS OF BIRDS

Species	Pesticide Concentration (ppm)	% Survival	Significance Level	Source
Mallard duck	Control	57.0		Fink (1978b)
	10	48.0	0.0001	"
	300	57.6	NS	"
	1000	51.0	0.025	"
Bobwhite quail	Control	58.8		Fink (1978a)
	10	47.0	0.001	"
	300	37.0	0.001	"
	1000	41.5	0.001	"

*NS = Not significant.

Lower Aquatic Organisms

Vilkas (1976) reported the 48-hour LC_{50} for technical metolachlor to be 25.1 ppm Daphnia magna. The 48-hr no-effect level was 5.6 ppm. Based on these data, the EPA (1980) concluded that metolachlor was slightly toxic to aquatic invertebrates.

Soil Microorganisms

Ercegovich et al. (1978a) studied the effect of metolachlor on 27 species of microorganisms, including the genera Bacillus, Cellulomonas, Cytophaga, Flavobacterium, Pseudomonas, Achromobacter, Aspergillus, Chaetomium, Fusarium, Penicillium, and Trichoderma. At 5 ppm metolachlor, the polulation growth of 6 of 27 species was inhibited; at 25 ppm, 9 of 27 species were inhibited, with a static (but not cidal) effect shown at both concentrations. The EPA (1980) concluded that if metolachlor was applied as directed at 1-3 lb a.i./acre, slight inhibitory/static effects would be expected. The adverse effects would lessen with time, and populations would be expected to recover.

Ercegovich et al. (1978b) studied the effect of 5, 25, and 125 ppm concentrations on nitrification rates in Morrison sandy loam and Hagerstown silt loam. Morrison sandy loam showed no effects at any concentrations evaluated, and the Hagerstown silt loam showed inhibition only at 125 ppm, which lasted for a 7-week period (recovery began at week 8).

Bioaccumulation

One study by Elleghausen (1977) indicated that algae and Daphnia accumulated 10.4 and 0.60 ppm when exposed to 0.1 ppm metolachlor. An 8-hour period of depuration was needed for a 50% loss of the accumulated metolachlor. Catfish (also exposed to 0.1 ppm) accumulated 1.20 ppm in a 96-hour exposure.

Smith (1977) and Barrows (1974) measured the bioaccumulation of metolachlor in fish. The EPA (1980) concluded that both studies indicate that metolachlor accumulates in fish.

Toxicity Data Evaluation

EPA, in its registration standards review of special studies using metolachlor, stated that "although no positive evidence of general chronic, teratogenic, fetotoxic, oncogenic, or mutagenic effects has so far been presented, the available information is presently insufficient to satisfy all the agency's requirements for the study of chronic effects."

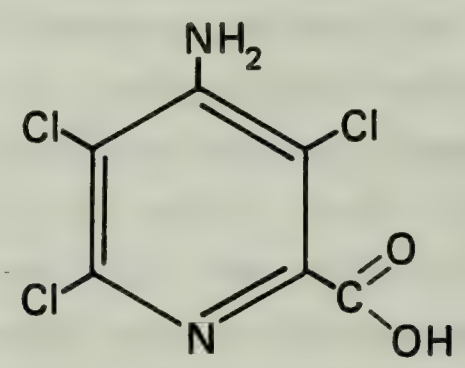
Specifically, gaps identified in EPA's Registration Standard included a mammalian oncogenicity study (other than one using a mouse), a rat chronic feeding study, a mammalian reproduction study, and a mammalian teratology study (other than one using a rat). The registrant has submitted new data to satisfy these requirements; these studies are currently being reviewed by EPA.

Several studies on metolachlor were conducted by IBT. EPA decided that portions of two studies done by IBT on chronic oral effects and reproductive effects are valid and could be used as supplemental information. Both of these studies have been replaced by Ciba-Geigy. Several chronic studies and one reproductive study done by other laboratories already exist in EPA files. A carcinogenicity study done by IBT is still under validation review.

L. PICLORAM

1. INTRODUCTION

Picloram is the common name for the herbicide 4-amino-3,5,6-trichloropicolinic acid produced by Dow Chemical Company. It is also called Amdon® (TRW, 1981), Borlin®, and Borolin®, and Tordon®. Tordon 10K® and Tordon 22K® contain picloram only, while Tordon 101® contains 2,4-D, and Tordon 155® contains 2,4,5-T. Numerous other formulations are available. Picloram can be applied as a spray solution to leaves or stems, as pellets to the ground, or as a liquid injected into the tree or painted on cut surfaces (TRW, 1981). The structure of picloram is



Other relevant physical and chemical parameters are presented below in the discussion of the fate of picloram in soil and water.

2. TOXICITY

Acute Toxicity

Acute toxicity tests indicate that picloram has a low order of toxicity. The LD_{50} values are 8200 mg/kg, 3000 mg/kg, and 2000 mg/kg in rats, guinea pigs, and rabbits, respectively. In mice, the LD_{50} values range from 2000 to 4000 mg/kg (Mullison, 1979). Tordon 101® (a formulation of picloram and 2,4-D) has an LD_{50} value of 3800 mg/kg in female rats (Lynn, 1965). In rabbits, the dermal LD_{50} value is >4000 mg/kg (U.S. Forest Service, 1974). NRCC (1974) reports that picloram does not appear to present an acute inhalation hazard, since no adverse effects

were seen in albino rats exposed for 7 hours to a saturated atmosphere of the potassium salt of picloram. Additionally, no adverse effects were seen after a 7-hour exposure to air that was bubbled through a solution of Tordon 22K® (Lynn, 1965).

No skin irritation was observed when various concentrations of picloram were applied to the skin of rabbits for several days. Rabbits exposed dermally to undiluted picloram for 9 days exhibited only slight exfoliation and hyperemia (U.S. Forest Service, 1974). Slight to moderate conjunctival irritation resulted when the eyes of albino rabbits were exposed to undiluted picloram. These effects cleared within a week (NRCC, 1974). In addition, when undiluted picloram was applied directly to the conjunctival sac of these animals, a slight redness and corneal cloudiness appeared, but cleared within 1 to 2 days (Lynn, 1965).

Subacute Toxicity

The results of a 90-day feeding study with rats showed no apparent adverse effects and 75 mg/kg (Lynn, 1965). No toxic effects were observed at concentrations of up to 1000 ppm picloram. At 3000 ppm picloram, the liver-to-body weight ratio in females was increased. At 10,000 ppm picloram, slight to moderate, unspecified pathological changes of the liver and the kidney were noted. The organ-to-body weight ratios for the liver and kidney were also significantly increased (McCollister and Lang, 1969). In a 6-month feeding study in which Sprague-Dawley rats were fed 100 mg/kg of the potassium salt of picloram, no adverse effects were observed. At 1000 mg/kg, females showed a reduced growth rate and a significant increase in organ-to-body weight ratios for the kidney, liver, and suprarenals. In males fed 1000 mg/kg, a significant increase was observed in organ-to-body weight ratios for kidney, liver, and testicles (Suschetet and Causeret, 1973; Suschetet et al., 1974). An 11-day study in which diluted picloram was applied nine times to the skin of rabbits resulted in slight exfoliation and hyperemia of the abdominal skin.

Mamalian Metabolism and Uptake

Picloram appears to be rapidly excreted from the mammalian system and does not accumulate in tissues (McCollister and Leng, 1969). These authors report that 90% of carboxyl-^{14}C-labeled picloram fed to dogs at a concentration of 97 ppm was excreted unchanged in the urine within 2 days.

Special Studies

Carcinogenicity The major study available on the carcinogenicity of picloram was conducted the the NCI (1978) on rats and mice. In this study, 50 male and 50 female Osborne-Mendel rats or B6C3F1 mice were administered technical grade picloram at the maximally tolerated dose and at one-half this amount for 80 weeks. These dosages (time-weighted) were 7437 and 14,875 ppm for rats and 2531 and 5062 ppm for mice. A disproportionately small number of controls (10 rats and 10 mice) were used in this study.

The authors concluded that picloram was not carcinogenic for B6C3F1 mice or male Osborne-Mendel rats, based on a statistically insignificant incidence of malignant tumors. A statistically significant increase in neoplastic nodules of the liver (benign tumors) was observed in rats, along with treatment-related lesions of the liver diagnosed as foci of cellular alteration. In addition, the results indicated a relatively high but statistically insignificant incidence of follicular hyperplasia, C-cell hyperplasia, and C-cell adenoma of the thyroid in rats of both sexes.

This study has generated considerable discussion. Part of this discussion has concerned the significance of the benign neoplastic nodules and the lesions diagnosed as foci of cellular alteration, in relation to the potential for malignancy. The lesions of cellular alteration have been commonly observed in association with the induction of neoplastic nodules and hepatocellular carcinomas in rats (Squires and Levitt, 1975).

The biological nature and significance of neoplastic nodules in rodents is currently a subject of controversy. Hirota and Williams (1979) found

that liver neoplastic nodules in rodents did not regress upon discontinuation of the inducer carcinogen (fluorenylacetamide), and new nodules grew. However, no direct evidence was found for the progression of the nodules to carcinomas. The Occupational Safety and Health Administration (OSHA), on the other hand, received testimony from a large number of pathologists who urged the agency not to differentiate between benign and malignant tumors when addressing carcinogenic potential. Each of these pathologists cited a number of carcinomas with benign (or apparently benign) precursors. Based on this testimony, OSHA decided not to draw a distinction between benign and malignant tumors in a carcinogenicity study unless that study could demonstrate no evidence of progression to malignancy, according to a set of criteria outlined by OSHA.

The National Cancer Institute (NCI) study has also come under question because of a reexamination of this study by Melvin Reuber (1981), a toxicologist at NCI. Based on his reexamination, Reuber concludes that picloram is carcinogenic. Reuber reviewed the histological sections for the rat and the mouse studies, and concluded that total neoplasms (both benign and malignant) at all sites were increased for both male and female rats in both the high and low dose treatment groups. Carcinomas of the adrenal, thyroid, and pituitary glands were increased in male and female rats, as were neoplasms of the liver and female reproductive organs. In regard to mice, Reuber concludes that neoplams of the spleen were increased in both male and female mice.

Reuber's findings of a significant increase in the number of neoplasms at all sites in rats is partially a result of his comparing tumor incidences in picloram-treated rats with pooled controls rather than matched controls. NCI generally runs carcinogenicity tests for several chemicals at the same time. Each study has a group of animals that are matched (for age, sex, etc.) to animals in the treatment group. Reuber pooled all the control animals from the various studies rather than comparing the results for the smaller number of matched control rats. Although this results in a firmer statistical basis for interpretation, it raises questions regarding the effect of the differences in age, sex, and other

characteristics of control rats, along with the possible difference in laboratory conditions and handling.

In another study, picloram caused proliferative lesions in endocrine organs when picloram was administered in the diet at the maximally tolerated dose and at half that amount. However, the author stated that the increase in these lesions was small and inconsistent and not indicative of carcinogenicity (Robens, 1978; abstract only). In a study by Dow Chemical, U.S.A., rats were given dietary doses of 15, 50, and 150 mg/kg for 2 years. No increase in the incidence of tumors was noted over control levels (Lynn, 1965; McCollister and Leng, 1969).

The data presented above do not allow a definitive statement regarding the potential carcinogenicity of picloram. The histological slides from the NCI study must be made available for further examination, and/or another study with a firmer statistical foundation must be conducted. Most reviewers of the NCI study, however, (EPA, for example) agree with NCI's finding that picloram causes benign neoplastic nodules. It seems advisable to adopt OSHA's position and suspect picloram as a possible carcinogen until it can be shown that these nodules do not progress to carcinomas.

Teratogenicity/Reproduction Limited data suggest that picloram does not cause teratogenic or adverse reproductive effects. In a study by Dow Chemical U.S.A., three generations of rats were given 3000 ppm picloram in their diet. No adverse effects were noted with respect to fertility, viability, gestation, body weight, lactation, or incidence of terata. No data were provided. (McCollister and Leng, 1969) The same study noted no effect on fertility or litter size when mice were given a dietary dose of 1000 ppm picloram for 4 days prior and 14 days subsequent to mating.

Thompson et al. (1972) found no terata or adverse effects on neonatal development in rats given daily oral doses of 0, 500, 750, or 1000 mg/kg/day of picloram on days 6 to 15 of gestation. Maternal deaths were noted at higher doses (5 at 750 mg/kg; 9 at 1000 mg/kg) between

days 7 and 17 of gestation. Mild diarrhea and hyperesthesia (excessive sensitivity of the skin) were also noted at higher doses. At 500 mg/kg, no overt signs of toxicity were noted. No effects on maternal weight gain, litter size, pup weights, or number of implantations, corpora lutea, and resorptions per dam were seen at any dose. Delivery and lactation were normal, as were survival and development of the pups. When fetuses were taken by Caesarean section on day 20, an increase in unossified fifth sternebrae was noted. However, this effect can be considered transient, since weanlings delivered by normal births had fully ossified sternebrae.

Mutagenicity As shown in Table II-43, most tests show no mutagenic activity of picloram.

Summary An evaluation of important toxicity data is provided at the end of this chapter.

3. MOBILITY AND PERSISTENCE

Fate in Soil

Picloram is generally considered to be relatively mobile in most soils (TRW, 1981; Arthur D. Little, Inc., 1979; USDA, 1973). The mobility of picloram can be attributed to its solubility (430 mg/l for picloram; 40% w/w for the potassium salt of picloram), to the low initial adsorption to soil colloids, and to the slow breakdown of picloram by soil microorganisms (NRCC, 1974).

In a review of leaching studies involving 24 soils from a number of agricultural states and provinces of Canada, the National Research Council of Canada (1974) stated that picloram penetrated to a depth of 30 cm or more in 83% of the soils, to a depth of 60 cm or more in 58% of the soils, and to a depth of 90 cm or more in 25% of the samples. (Application rates ranged from 0.23 to 10.08 kg/ha). Helling (1971a) classified picloram as "highly mobile" in a study of 40 pesticides. Phillips and Feltner (1972) showed picloram to be highly mobile, using 3.36 kg/ha applied to a Kansas sandy clay loam. After 3 years, the concentrations of picloram detected were 229, 279, 278, 31, and 43 ppb in the 0-15, 15-30, 30-60, 60-75, and 75-120 cm layers of the soil, respectively. Bowes (1972) also found very

TABLE II-43

MUTAGENICITY TESTS: PICLORAM

Test System	Result	Comments	Reference
rat bone marrow *in vivo*	-	200 mg orally	Mensik et al., Dow Report (no (date available)
Aspergillus nidulans			
Point mutation	-		Aulicino et al. (1976) and
Mitotic crossing over	-		Bignami et al. (1977)
Mitotic nondisjunction	-		
Streptomyces coelicolor A3	+		Carere et al. (1978)
	+		Carere et al. (1978)
	+		Torracca et al. (1976)
Ames/*Salmonella*	-	5µg/plate	Anderson et al. (1972)
	-		Carere et al. (1978)
	-		Carere et al. (1978)
	-		Torracca et al. (1976)
rII mutants of T_4 bacteriophage/*E. coli*	-	500µg	Anderson et al. (1972)

high mobility after applying 4.48 kg/ha picloram to a heavy clay and a sandy loam in Saskatchewan. He found an average of 149, 55.5, 114.5, 128, and 30 ppb in the 0-15, 15-30, 30-60, 60-75, and 75-120 cm layers of the soil.

The extent to which picloram moved in the soil was assessed along a powerline right-of-way in the Pacific Northwest. Picloram moved down to 30 cm, although most of it stayed above 15 cm (USDA, 1977). EPA (no publication date provided [f]) states that picloram remains in the upper 20-30 cm depth in most soils, except those of a sandy nature. Norris et al. (1976a) found that when picloram was applied to a southern Oregon hillside pasture, most of the herbicide remained in the upper 6 inches (15.24 cm).

Because of its persistence, picloram can appear in soil leachate for a considerable length of time (Arthur D. Little, Inc., 1979). Nine to 12 months after treatment, picloram was found at levels of 1 to 4 ppb in leachate water (Arthur D. Little, Inc., 1979). In a study by Glass and Edwards (1974), the first detection of picloram (1 ppb) at a depth of 240 cm was found one year after application. After two years, picloram was still detectable at 0.5 ppb. Helling (1971b, c) found no correlation between the mobility of picloram and the properties of 14 different soils (e.g., pH, organic matter) due to the low adsorption of picloram in these soils. Mobility of picloram was found to be correlated only with the amount of water flowing through these soils.

TRW (1981), on the other hand, reviewed the literature and found that adsorption (and thus mobility) is a function of pH, application rate, soil type, and formulation, as well as water flux. Organic matter is considered the most important of these factors. Adsorption readily occurs in soils containing a high organic content (Norris, 1970b) high concentrations of humic acid (Khan, 1973), and significant amounts of the hydrated oxides of aluminum and iron (Hamaker et al., 1963). Adsorption increases with increasing acidity, but is minimal in alkaline or neutral soils (Youngson et al., 1967; Biggar and Cheung, 1973; Farmer and Aochi, 1974; Grover, 1971; McCall et al., 1972). As would be expected, given the various adsorption capacities of different soil

types, the movement of picloram is greatest in soils that are poor in organic matter or in sandy, light-textured soils (Mullison, 1979; NRCC, 1974). The triisopropanolamine salt of picloram was found to be less mobile than the potassium salt (Hunter and Stobbe, 1982). Bovey and Scifres (1971) found that the leaching rates of esters and salts of picloram were similar, and only the acid form was found below 5 cm.

Runoff studies have indicated that picloram is likely to move in water as it flows over the soil (TRW, 1981; Arthur D. Little, Inc., 1979). NRCC (1974) states, however, that runoff is likely to remove only small quantities of picloram from the soil. Norris (1969) concludes that picloram will be found in runoff when rainstorms are sufficiently intense to cause overland flow rather than infiltration.

Norris (1969) found that when picloram was applied to three forest plots in Oregon and Washington at rates of 0.5 to 1.0 lb/acres, the maximum concentration detected in the runoff was 20-78 ppb. Bovey et al. (1967) found that the potential for high concentrations of picloram in the runoff increases as the time between the application and the first rainfall decreases. Immediately after applying a 1:1 mixture of triethylamine salt at the rate of 1:12 kg/ha on grassland watersheds, heavy rainfall occurred, resulting in maximum concentrations in the surface runoff of 400-800 ppb. Davidson and Chang (1979) found similar results when picloram pellets were applied at a rate of 9.0 kg/ha to 4.5% of a forested Arizona watershed. The maximum concentration detected was 370 ppb, after a storm of 72 mm.

Other studies do not indicate such high concentrations of picloram in the runoff. From a forest plot in Ontario, Canada, where picloram had been applied at a rate of of 0.9 kg/ha, the concentration in the drainage was 38 ppb after 1 day, 26 ppb after 7 weeks, and 1 ppb after 1 year. Baur et al. (1972) determined that 10-12 weeks after the application of 1.12 kg/ha to 8 ha plots, about 10 ppb was found in runoff water adjacent to the plots. Water sampled 1.2 km from the plots after 8 days contained 1 ppb.

Conflicting results are documented for the effect of formulations in the form of pellets and sprays. Bovey et. al. (1978) stated that the potassium salt generated similar concentrations of picloram whether applied as an aqueous spray or a pellet. A later study by Burnett and Richardson (1980) showed considerable difference in the rate of runoff loss for sprays and granules applied at 2 kg/ha to large watersheds in Texas. They found a large initial concentration (112 ppb) immediately after application of the spray, but not after application of the starch xanthate granules (6 ppb immediately after application). Runoff loss from the slow-release granules continued to increase for several weeks while those from the sprayed watershed dropped to 0.1 ppb. Runoff concentrations stayed at 20 ppb for 14 weeks, then dropped to 4 ppb after 8 months. After 9 rainfall events, a total of 2.5% of the sprayed picloram had been lost, while only 1.5% of the slow-release picloram had been lost. Lower vertical mobility of the granules may also be indicated, since picloram concentrations in the upper 15 cm of soil were greater for the granules than for the spray.

Picloram is well-documented as being moderately to highly persistent, with half-lives of 1 to 13 or more months (Mitchell, 1969; Schlapfer, 1977; EPA registration files, no date; Goring et al., 1965; Altom and Stritzke, 1973; Hamaker et al., 1967.

The persistence of picloram is a function of soil type, moisture, and temperature. It is very persistent in cold, dry climates, and in clay and sandy loam soils having a low percentage of organic matter. (Caro et al., 1974; Merkle et al., 1967; Hunter and Stobbe, 1972; Herr et al., 1966b). Additionally, dissipation is very slow under conditions of low soil moisture (Hunter and Stobbe, 1972) and high pH (Youngson et al., 1967). The effect of organic matter is unclear (Arthur D. Little, Inc., 1979). Barnside et al. (1971) and Herr et al. (1966) state that persistence increases with organic matter, while Helling (1971a,b,c) and Merkle et al. (1973) state that it decreases with organic matter.

Picloram does not serve as a good energy source for microorganisms, although it is cometabolized with other energy sources, and amounts degraded may be small, even under the most favorable conditions.

The mechanism of microbial degradation is unknown, and it may vary from organism to organism. It is believed, however, to be a decarboxylation and ring cleavage (TRW, 1981). Merkle, et al. (1974) used lab studies to show that the ring-labeled picloram is degraded to produce CO_2 at approximately the same rate at which the carboxyl-labeled carbon reacts. The by-products identified were 4-amino-2,3,4-trichloropyridine and 6-hydroxy-3,5-dichloro-4-aminopiclonic acid.

Residues and Persistence in Water

Initial picloram residues in water have been shown to be highly variable. Residual concentrations decrease rapidly, however, and picloram appears to be less persistent in water than in soil. The routes of loss of picloram from water are unclear.

USDOE (1980) documents two studies (giving no primary sources) in which picloram was applied aerially at a rate of 1 lb/acre to transmission line rights-of-ways. In the first study, involving a segment in the Cascade Range in Oregon, residual concentrations peaked at 15 ppb 30 minutes after application to a site approximately 350 feet from the stream sampling area. After 1 hour, the concentration was below the detection level of 3 ppb, and picloram was not detected in the subsequent 5-month period. In the same study, also at 1 lb/acre, no residues were detected (limit, 2 ppb) for 9 months after application, although equipment failure resulted in no monitoring in the first 48 hours. It was found later that direct application to the stream had occurred. During the 9 months, 70 inches of precipitation had fallen in this area on the Oregon coast, so it is likely that picloram residues were rapidly diluted and flushed from the area.

Similar results have been found in other stream studies. Five months after 1 kg/ha picloram was applied to plots located near the head of a small stream, Haas et al. (1971) found no detectable picloram at distances of 0, 0.8, and 1.6 km from the plots, even though runoff water contained a maximum of 29 ppb during that time. In another study in which runoff water entering a creek contained 13 ppb

picloram, concentrations in the creek were 0.4 ppb or less (no time given) (Baur et al., 1972).

Standing water may contain higher concentrations of picloram. Haas et al. (1971) applied picloram directly to a livestock pond at a rate of 1.12 kg/ha and found an initial concentration of 500 ppb, which dropped to about 5 ppb after 100 days and remained at that level for another 100 days. Picloram was not detectable (limit, 1 ppb) 1 year later. In the same study, a more complicated system was examined in which one large pond received picloram from direct application (at 1.12 kg/ha) and from runoff from surrounding grassland. The initial concentration in this pond was as high as 1000 ppb immediately after application. After a heavy rainfall this pond overflowed into 2 smaller ponds, which were found to contain 10 to 20 ppb immediately after overflow occurred. After 100 days, however, concentrations in all ponds decreased to 1 to 2 ppb. The author found from both of these studies that the loss of picloram was concentration-dependent, with an initial rate of loss of 14% to 18% per day in the first 100 days, and a subsequent rate of loss of 1% per day in the next 100 days.

Picloram is not subject to significant microbial or chemical degradation in water; photodegradation is considered the major degradation route (NRCC, 1974; TRW, 1984). However, photodegradation is usually significant only in the upper surface layers of water. This is demonstrated by the photolysis half-lives of picloram, which range from 5 days in 1-inch deep containers to 60 days in 12-foot deep non-circulating containers. (Hedelund and Youngson, 1972). Thus, primary routes of loss remain unclear.

Indicators of Potential Ground Water Contamination

Table II-44 provides information on parameters associated with the mobility of picloram. These parameters, and their associated thresholds, have been suggested by EPA for use in assessing the potential for pesticide contamination of ground water. A discussion of these parameters and thresholds, and the methods for arriving at designated values for individual herbicides, is presented in the main body of the

report as part of the discussion of the fate of herbicides in the environment.

TABLE II-44

INDICATORS OF POTENTIAL GROUND WATER CONTAMINATION: PICLORAM

Indicator	Value for Picloram	Threshold
Solubility	430 ppm at 25°C (potassium salt: highly soluble)	>30 ppm
K_{oc}	13	<300-500
Speciation at pH 5	Anionic	Anionic (negatively charged)
Hydrolysis half-life	Stable	>6 months
Photolysis half-life	<1 week	>3 days
Vapor pressure	6.16×10^{-7} mm Hg at 20°C	$<10^{-2}$ mm Hg

* ND = no data.

4. TOXICITY TO NON-TARGET ORGANISMS

Birds

Kenaga (1969), Mullison (1972), Bovey and Scifres (1971), and others concluded that picloram had low toxicity to birds. Kenaga (1969) studied the effects of picloram on three generations of Japanese quail by supplying 1000 ppm to their feed. The author concluded that there was no effect on mortality, egg production, or fertility. The author also determined the LC_{50} for bobwhite quail and mallard ducks to be 23,000 and 385,000 ppm, respectively. Norris (1976) reported an LD_{50} for birds to be greater than 2000 mg/kg, and the author noted that 1000 mg/kg produced no effect. Tucker and Crabtree (1970) reported

TABLE II-45

TOXICITY OF PICLORAM TO BIRDS

Species	Formulation	Comments	Source
Mallard duck	90.5% a.i.	LD_{50} >2000 mg/kg	Tucker and Crabtree (1970)
	Tordon 22K	LD_{50} >2000 mg/kg	"
	Not given	LC_{50} >5000 ppm	Hill et al., (1975) and Heath et al., (1972)
	Acid equivalent	500-10,000 mg/kg in feed for 5 days produced no mortality	Kenaga (1969)
Bobwhite quail	Acid equivalent	LC_{50} = 2336 mg/kg	"
Bobwhite quail, 5-7 day old chicks	Acid equivalent	LC_{50} = 10,000 mg/kg	"
Bobwhite quail	Not given	LC_{50} > 5000 ppm	Heath et al., (1972) and Hill et al., (1975)
Japanese quail	Not given	LC_{50} > 5000 ppm	"
Japanese quail	Acid equivalent	100 mg/kg in feed produced no effect on plumage, feathering, fertility, egg production, hatchability, mortality, or weight for 14-day exposure	Kenaga (1969)

TABLE II-45 (CONTINUED)

TOXICITY OF PICLORAM TO BIRDS

Species	Formulation	Comments	Source
Japanese quail	Acid equivalent	1000 mg/kg for 14-day exposure produced no effect on egg production, body weight, or adult mortality. Egg fertility reduced 55%; egg hatchability reduced first week but not second week of treatment. Hatchability and fertility normal after first week.	Kenaga (1969)
Japanese quail, 5-7 day old chicks	Acid equivalent	100, 500, and 1000 mg/kg given in reproductive, 3-generation study. F_0 generation fed 20 weeks, F_1 generation fed 12 weeks, F_2 generation fed 3 weeks. No significant reduction in food consumption, egg production, fertility, hatchability, survival,or weight gain. No adverse symptoms noted when diet returned to normal.	"
Pheasant	Not given	LC_{50} >5000 ppm	Hill (1975) and Heath (1972)
Pheasant	90.5% a.i.	LC_{50} >2000 mg/kg	Tucker and Crabtree (1970)

an LD_{50} value for mallards and pheasants to greater than 2000 mg/kg. Heath et al. (1972) and Hill et al. (1975) reported LC_{50} values for Japanese and bobwhite quail, ring-necked pheasants, and mallard ducks to be greater than 5000 ppm. These data are summarized in Table II-45.

Fish

The toxicity of picloram is shown in Table II-46. Arthur D. Little, Inc. (1979) concluded that picloram was toxic to fish, based on the fact that 96-hr LC_{50} values ranged from 1.55 to 26.0 ppm, depending on the species of fish and the formulation of picloram. A review by the USDA (1973) stated that the isooctyl ester would be toxic to sensitive species. TRW (1981), on the other hand, stated that picloram and its salts were low in toxicity to fish, based on data provided by EPA (publication date not provided [f]), and Sargent et al. (1971). Data they cited was primarily from 24-hr toxicity tests, which may explain the difference between the TRW and Arthur D. Little, Inc., conclusions.

Kenaga (1969) stated that a field application of 3 lb/acre of picloram was not likely to result in contamination greater than 1 ppm, due to dilution, adsorption, and degradation.

A study by Woodward (1979) concluded that picloram reduced growth in cutthroat fry at concentrations above 0.61 ppm, and increased fry mortality at concentrations greater than 1.3 ppm. No adverse effects were noted at concentrations below 0.290 ppm. Lorz (1979) reported the effects of picloram on Coho salmon. In salt water, 0.29-19.8 ppm for 144 hrs produced little effect, but 0.29-.62 ppm produced an unexplained mortality of 75%. The effect of Tordon 101® at 1.35 and 1.8 ppm had little or no effect on seaward migration of the Coho salmon.

Lower Aquatic Organisms

Hardy (1966) studied the effect of 1 ppm of the potassium salt of picloram on a food chain of algae, daphnids, and fish. The author found that the algal growth was not retarded, and that the daphnids and fish appeared to behave and reproduce normally. Table II-47 sum-

TABLE II-46

TOXICITY OF PICLORAM TO FISH

Formulation	Species	Test	Result (ppm)	Source
Technical grade	Cutthroat	96-hr TL_m	3.45-8.60	Woodward (1976)
	Lake trout	96-hr TL_m	1.55-4.95	"
Tordon 22K	Rainbow trout	48-hr LC_{50}	31.0	Fogels and Sprague (1972)
Tordon 22K	Rainbow trout	96-hr "	26.0	"
Potassium salt	Rasbora heteromorpha	48-hr "	11.00	Alabaster (1969)
Potassium salt	Harlequin fish	24-hr "	66	Kearney and Kaufman (1975)
Potassium salt	Channel catfish	24-hr "	41	"
Potassium salt	Bluegill	24-hr "	69	"
Acid	Fathead minnow	24-hr "	64-135	"
	Rainbow trout	24-hr "	34-250	"
	Rainbow trout	48-hr "	2.5	"
	Green sunfish	24-hr "	150-240	"
	Brown trout	24-hr "	230-240	"
	Brook trout	24-hr "	240-420	"
	Black bullhead	24-hr "	420	"
	Bass	24-hr "	19.7	"
	Coho salmon	24-hr "	29	"
Triisopropanolamine salt	Rainbow trout	24-hr "	279	"
Triethylamine salt	Rainbow trout	24 hr "	43.4	"
	Channel catfish	24 hr "	70.5	"
	Goldfish	24 hr	90.6	"

TABLE II-47

TOXICITY OF PICLORAM TO LOWER AQUATIC ORGANISMS

Formulation	Species	Comments	Source
Tordon 22K	Phytoplankton mix	4-hr exposure[1] produced 8.4% decrease in productivity	Butler (1965)
Tordon 101[2]	Phytoplankton mix	4-hr exposure[1] produced no effect on productivity	"
Tordon 101[2]	Oyster	1 ppm for 96 hours produced no effect on shell growth	"
Tordon 101[2]	Brown shrimp	1 ppm for 48 hours produced no effect on survival	"
Not stated	Molluscs	380 ppm produced no effect, but 530 ppm produced 100% mortality	"
Not stated	Stonefly nymphs	24-hr LC_{50} = 120 ppm	Sanders and Cope (1968)
Not stated	Amphipods	48-hr LC_{50} = 48 ppm	Sanders (1969)
Not stated	Daphnia	380 ppm produced no effect, but 530 ppm produced 95% mortality	Lynn (1965) and House, House et al. (1967)

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[1] No concentration cited.

[2] Includes 2,4-D.

marizes the data on the effect of picloram on lower aquatic organisms.

Livestock

Mullison (1979), EPA (publication date not provided [f]), NRCC (1974), Norris (1976), and others stated that picloram had a low toxicity to warm-blooded animals. Williams (1971b) fed 23 mg/kg/day to cattle, and 100 mg/kg/day to sheep for 1 month and observed no adverse effects.

Lynn (1965) reported that sheep showed no adverse effects when fed the potassium salt formulation (25% active ingredient) at a rate of 4650 mg/kg, but the Tordon 101® formulation produced toxic effects and subsequent death in 3 days at 2530 mg/kg. Cattle appear to be a bit more sensitive, with 1900 mg/kg representing the toxic dose. It should be noted that the doses used in this study are very large in comparison with those that could result from exposure. Mullison (1979) reported LD_{50} values of 6000 mg/kg for chicks, greater than 1000 mg/kg for sheep, and greater than 750 mg/kg for cattle.

Bees

Johansen (1980) and the University of California (1975) reported that picloram showed a low toxicity to bees. Johansen (1980) reported the LD_{50} values for both picloram and Tordon (formulation not given) to be 15 µg/bee. Morton et al. (1972) found that newly emerged honeybees fed concentrations of 0, 10, 100, and 1000 ppm by weight in 60% sucrose showed no reduction in half-life. The authors actually reported an increase in half-life of bees fed 100 and 1000 ppm. Moffett et al. (1972) concluded that Tordon 22K® and Tordon 212® applied at a rate of 4 lb a.i./acre were non-toxic to bees confined in a 20 gal/acre water carrier.

Soil Microorganisms

The EPA (publication date not provided [f]), Mullison (1979), and NRCC (1974) stated that picloram was low in toxicity to soil microorganisms. Goring (1971) noted that it was broken down in soil and by pure cultures of a variety of microorganisms. In another study, Goring

et al. (1967) subjected 46 different common microorganisms to concentrations of between 0 and 1000 ppm, and found that it did not retard growth or development of any of them except Thiobacillus thiooxidans, which was inhibited at 1000 ppm but not at 100 ppm. The authors also concluded that rates of carbon dioxide evolution and urea hydrolysis were unaffected. At 1000 ppm nitrification of ammonium ions to nitrite ions was partially inhibited, but not at 100 ppm. Tu and Bollen (1969) found little effect up to 1000 ppm on ammonification, nitrification, sulfur oxidation, and organic decomposition.

Arnold et al. (1966) found that growth of Aspergillus niger was not depressed by the addition of 0.4-5.0 ppm picloram in nutrient solution, although it did accumulate in the mycelia. A study by Hameed and Foy (1974) assessed the effect of 1 to 1000 ppm picloram on five species of soil fungi (Trichoderma viride, Fusarium oxysporum, Helminthosporium victoriae, Penicillium lanosum, and Aspergillus flaves). All species grew, but were not able to utilize picloram as a sole source of carbon and nitrogen.

Hardy (1966) and Elder et al. (1970) investigated the effect of picloram on algae. The first study found that 1 ppm picloram in water had no effect on algae, and the second study found that picloram had a low toxicity to many fresh water and marine algae species at concentrations approaching its maximum solubility in water.

Bioaccumulation

A number of studies have shown that picloram does not bioaccumulate in animals. When steer were fed 200-1600 ppm picloram, a maximum of 0.3 ppm was found in muscle and fat, and up to 18 ppm in kidneys. The concentration fell to less than 0.1 ppm within 3 days of withdrawing the picloram from the diet (NRCC, 1974). McCollister and Lang (1969) and Norris (1971) stated that cattle and other mammals eliminated 98% of ingested picloram as an unchanged compound in the urine. The EPA (publication date not provided [f]), and Hardy (1966) found that picloram did not accumulate in aquatic food webs or chains.

Studies by Arnold et al. (1966) and Hameed and Foy (1974) did indicate that picloram was accumulated in the mycelia of a variety of fungal species.

Toxicity Data Evaluation

The evaluation of carcinogenicity data for picloram has been stated on the previous page: The data do not allow a definitive statement. Insufficient data are available to indicate with any certainty that picloram does not cause teratogenic effects. Although most of the tests on mutagenicity show no effect, an insufficient number of reliable tests have been conducted to draw a definitive conclusion.

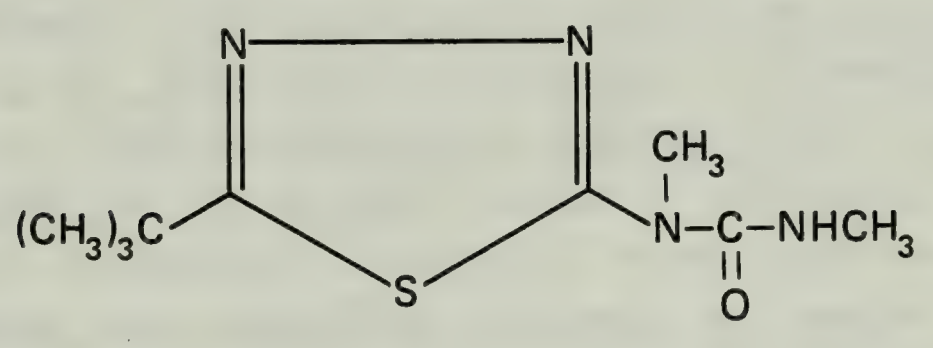
Dow Chemical U.S.A. has replaced all studies conducted by IBT on picloram; namely a teratogenicity test using the mouse, a skin-patch test using humans, and two chronic oral studies using the dog and the rat. Two teratology studies and one chronic study done by other laboratories already existed in EPA files.

A registration standard on picloram is currently being prepared by EPA. Data gaps to be filled will be identified in that document.

M. TEBUTHIURON

1. INTRODUCTION

Tebuthiuron is the common name for a substituted urea herbicide, 1-(5-tert.-butyl-1,3,4-thiadiazol-2-41)-1,3-dimethylurea, manufactured by Elanco Products Company, a Division of Eli Lilly and Company. It is also called Graslan®, Brulan®, Tiurolan®, EL-103®, Preflan®, Perfmide®, Tebulan®, Prefmid®, and Spike®. Its chemical structure is shown below.



Relevant physical and chemical characteristics are presented below in the discussion of tebuthiuron in soil.

2. TOXICITY

Acute Toxicity

Tebuthiuron shows moderate toxicity in acute tests. Lilly Research Laboratories (1982) states that the acute oral LD_{50} values are for the rat, mouse, and rabbit are 579 mg/kg, 644 mg/kg, and 286 mg/kg , respectively. This study also reports that rabbit tests showed no dermal irritation and only "slight transient" eye irritation. An information sheet by Elanco Products Company (1980) states that when rats were given a single oral dose of 500 mg Spike 80W® per kg of body weight, no effects were observed for a period of 14 days after treatment.

Mammalian Metabolism

Tebuthiuron appears to be rapidly eliminated. More than 85% of labeled tebuthiuron (single oral dose) was excreted in 96 hours in the rat, rabbit, mouse, dog, duck, and steer. (Lilly Research Laboratories, 1982).

Special Studies

The only available literature on the chronic toxicity of tebuthiuron is from a summary of information about Graslan® provided by Lilly Research Laboratories (1982). This report states that two multi-generation studies have shown no evidence of carcinogenicity, mutagenicity, teratogenicity, or impairment of reproductive performance. In a two- generation rat study, a minimal depression in bodyweight gain was observed in one sex, in one generation of rats at levels of 200 and 400 ppm. The no-effect level was found to be 100 ppm or 7 mg/kg/day tebuthiuron. Based on these studies, an acceptable daily intake was determined to be 0.0737 mg/kg/day tebuthiuron. Total maximum dietary intake was determined to be 0.0128 mg/kg/day, using established tolerances as a basis for intake levels.

Summary An evaluation of important toxicity data is provided at the end of this chapter.

3. MOBILITY AND PERSISTENCE

Fate in Soil and Water

Tebuthiuron appears to be a mobile and persistent herbicide. Manufacturer's information provided for Graslan® states that "GRASLAN pellets, deposited on the soil surface, are disintegrated by the first significant rainfall and the herbicide is moved into the soil. Subsequent rainfall moves the herbicide into the root zone where it is absorbed by the roots of woody plants. . . . The relatively long soil half-life and some vertical movement are considered necessary for the proposed use. . . ." Lilly Research Laboratories (1982) reports that tebuthiuron moved to a depth of 46-61 cm in one study, although other studies showed no tebuthiuron below 46 cm. In a study by Reed (1982), tebuthiuron moved to a depth

of 26 cm after 6 weeks in a silty clay, and to a depth of 30 cm (maximum depth sampled) after 6 weeks in a sandy loam and a sandy loam with high organic matter content. Baur (1978) found that 20 cm of rain distributed tebuthiuron throughout a 20 cm soil profile in both a clay loam and a sandy loam. In a soil thin-layer chromatography test, tebuthiuron was found to move with the water front in a sandy soil, although less movement was found in a loam and a silt loam (Chang and Stritzke, 1977).

Different results were found by C. D. Christensen et al. (1974), who found "little vertical movement" and "essentially no lateral movement" when tebuthiuron was applied along several miles of highway and railroad right-of-way in Massachusetts, New York, Pennsylvania, and Kentucky--even along slopes up to 20%. No further information was provided in a review of this study by Reed (1982).

Lilly Research Laboratories (1982) reports that wells in or adjacent to Graslan®-treated areas have contained no residues of tebuthiuron.

Contradictory information is available on the potential for movement via runoff. Bovey et al. (1978a) found up to 22 ppm in runoff when rainfall occurred shortly after application. Lilly Research Laboratories (1982) reports that no detectable residues were found in runoff water from an Arizona watershed, and a maximum of 0.18 mg/l was found in a catchment basin in a watershed in Texas. The maximum level was found after 7.1 inches fell in one day.

Tebuthiuron appears to be very persistent, with a half-life of 12 to 15 months in areas with 40 to 60 inches of rainfall (Thomson, 1975). Lilly Research Laboratories (1982) found second and third half-life values to be approximately 125 and 525 days, respectively, in areas with more than 30 inches of rainfall. Two studies found that tebuthiuron "may persist more than a year" (C. D. Christensen et al., 1974; Klingman and Ashton, 1975) (no further information provided). Precautions on the label (Elanco Products Co., 1980) include the statement that its "presence in the soil may prevent growth of other desirable vegetation for some years to come."

Degradation appears to be a microbial process, including ring cleavage to form volatile products, and demethylation of the urea moiety (Lilly Research Laboratories, 1982).

No information was found on residues or the persistence of tebuthiuron in surface waters.

Indicators of Potential Ground Water Contamination

Table II-48 provides information on parameters associated with the mobility of tebuthiuron. These parameters, and their associated thresholds, have been suggested by EPA for use in assessing the potential for pesticide contamination of ground water. A discussion of these parameters and thresholds, and the methods for arriving at designated values for individual herbicides, is presented in the main body of the report as part of the discussion of the fate of herbicides in the environment.

TABLE II-48

INDICATORS OF POTENTIAL GROUND WATER CONTAMINATION: TEBUTHIURON

Indicator	Value for Tebuthiuron	Threshold
Solubility	2300 ppm at 25°C	>30 ppm
K_{oc}	620	<300-500
Speciation at pH 5	Catonic (positively charged)	Anionic (negatively charged)
Hydrolysis half-life	ND*	>6 months
Photolysis half-life	stable in light	>3 days
Vapor pressure	non-volatile	$<10^{-2}$ mm Hg

* ND = no data.

4. TOXICITY TO NON-TARGET ORGANISMS

Birds

Insufficient information is available to evaluate the effect of tebuthiuron on birds. Low toxicity is suggested by a chronic one-generation reproduction study in which 100 ppm tebuthiuron caused no toxic symptoms in bobwhite quail, or mallards (Lilly Research Laboratories, 1982).

Fish and Lower Aquatic Organisms

Limited information suggests that tebuthiuron is not toxic to fish and lower aquatic organisms. A report by Lilly Research Laboratories (1982) found that the LC_{50} or EC_{50} for Daphnia magna, eastern oysters, pink shrimp, fiddler crabs, bluegill, and rainbow trout ranged from 48 mg/l to 320 mg/l. No symptoms of toxicity were observed in chronic embryo larvae studies with these organisms when exposed to 9.3 mg/l tebuthiuron. Thomson (1975) states that tebuthiuron is low in toxicity to fish.

Bees

Tebuthiuron appears to be non-toxic to bees. No greater mortality of honeybees was observed after they were sprayed with 30 g/l tebuthiuron than after they were sprayed with water (Lilly Research Laboratories, 1982).

Livestock

Tebuthiuron appears to be non-toxic to livestock. After being fed 10, 30, and 100 ppm tebuthiuron for 162 days, no toxic symptoms were noted, and weight gain was normal except for a slight reduction at the highest dose. In another study, Herford cows in Graslan®-treated pastures gained 108 lb/head, as compared to 44 lb/head in untreated pastures (Lilly Research Laboratories, 1982).

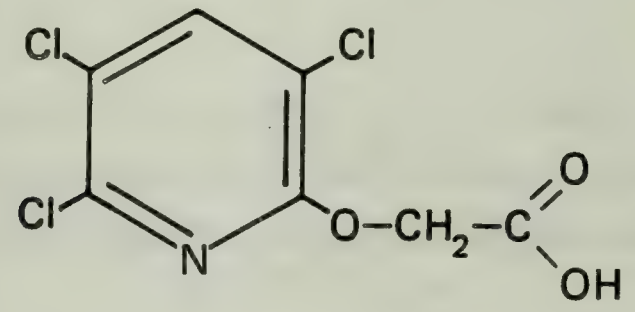
Toxicity Data Evaluation

More publicly available information is needed on tebuthiuron. However, since it was registered after the 1972 data requirements were in place, it can be assumed that the manufacturer conducted the complement of tests required by EPA at that time, and that the results were found to be acceptable by EPA. No tests were conducted by IBT.

N. TRICLOPYR

1. INTRODUCTION

Triclopyr is the common name for the herbicide ((3,5,6-trichloro-2-pyridyl)oxy) acetic acid, produced by Dow Chemical U.S.A. Triclopyr is available in two formulations: Garlon 3A®, a triethylamine (TEA) salt that is water-soluble and contains methonol, and Garlon 4®, an ethylene glycol butyl ether ester (EGBE) that is oil-soluble and water-emulsifiable (TRW, 1981). DOWCO 233 is an early name used to refer to triclopyr formulations (Thomson, 1975); however, no information is available on its properties. Garlon 3A® contains 3 lb triclopyr acid equivalent per gallon and Garlon 4® contains 4 lb triclopyr acid equivalent per gallon. Triclopyr is a picolinic acid derivative with the following structure:



Relevant physical and chemical properties are presented below in the discussion of the fate of triclopyr in soil and water.

2. TOXICITY

Acute Toxicity

Available information suggests that triclopyr can be considered slightly toxic, while Garlon® formulations have low toxicity. LD_{50} values range from 713 mg/kg for triclopyr (the active ingredient of Garlon) to 2830 mg/kg for Garlon 3A® (see Table II-49). The acute dermal LD_{50} in rats is greater than 4000 mg/kg, although repeated prolonged contact may cause irritation. An inhalation study showed that 100% of the test rats survived a 1-hour exposure to 3-20 dilutions of Garlon 3A® in air.

Transitory nasal irritation to rats was noted after a 4-hour exposure to Garlon 4® aerosol (Dow Chemical U.S.A.; 1981a).

While Garlon 4® is essentially non-irritating to eyes, Garlon 3A® can cause serious eye injury in humans. Permanent impairment of vision can result from exposure. Effects include severe conjuctival irritation, moderate internal redness, and moderate to severe corneal injury, which (tests show) is not healed after 21 days. Washing is not effective in preventing these effects (Dow Chemical U.S.A.; 1981a).

Special Studies

TRW (1981) cites the following information by Dow Chemical U.S.A. Triclopyr, administered at 30 mg/kg/day, was not carcinogenic in rats and mice. In dominant lethal and in host-mediated assays, triclopyr was not mutagenic. At 200 mg/kg/day, it exhibits a reproductive toxicity effect and is considered mildly fetotoxic.

Only one publicly available study was located. Moriya et al. (1983) found no evidence of mutagenicity in an Ames/Salmonella test system and an Escherichia coli WP2 test system.

Summary An evaluation of important toxicity data is provided at the end of this chapter.

TABLE II-49

ACUTE ORAL TOXICITY OF TRICLOPYR

	LD_{50} (mg/kg body weight)				
	Rats			Guinea	
Form	Male	Female	Rabbit	Pig	Source
Triclopyr, technical grade	713*				Thomson (1976)
Triclopyr	729	630	550	310	Dow Chem. U.S.A. (1979, 1981)
Garlon 3A	2830	2140			"
Garlon 4	2460	2140			"

* Unspecified sex.

3. MOBILITY AND PERSISTENCE

Fate in Soil

TRW (1981) states that triclopyr is considered a mobile herbicide, citing a review by EPA of registration file material. Manufacturer's information states that triclopyr does not readily adsorb to soil particles (Dow Chemical U.S.A. 1981). The mobility of triclopyr was studied by Hamaker (1977a) using a loam sand that was low in organic matter (0.62% organic carbon). Water was applied at a rate of 0.5 inches/day for 45 days. The results indicated that 75% to 80% of the triclopyr passed through a 12-inch column of soil between days 11 and 15. A degradation product, trichloropyridinol, was less mobile, requiring 13 inches of applied water to move through the column, as compared to 7.5 inches for the parent compound. McKellar (1977) studied the leaching potential in six soils (ranging from clays to loamy sands) under field conditions in six states. Garlon 3A® was applied at a rate of 3 gal/acre, and the rainfall was said to be normal, although the rates are not given. Small amounts (concentrations not given) of triclopyr and its degradation products were found at depths of 6 inches to 18 inches after 28 to 56 days. McKellar (1977) notes that the degradate trichloropyridinol is less mobile than triclopyr.

A somewhat confusing picture is presented in studies by Hamaker (1975) in his determination of distribution coefficients for the TEA salt in 12 soils that ranged in organic carbon content from 0.081% to 21.7%. K_{oc} values for triclopyr ranged from 12 to 78. Assuming a Massachusetts soil with 2% organic matter, this would give a K_d range of 0.24 (high mobility) to 1.56 (low mobility). As part of the same study, the mobility of the degradate trichloropyridinol was studied on three unspecified soils. The K_{oc}'s ranged from 114 to 156, which in a soil with 2% organic matter would give K_d's of 2.28 (low mobility) to 3.12 (nearly immobile).

Norris et al. (1976) studied the losses due to runoff. Triclopyr was applied as the TEA salt at the rate of 3 lb/acre to an area where 150 cm of rain fell in 9 months. Residues of 6 ppb and 1 ppb in runoff

water were measured 5 months and 9 months, respectively, after application.

TRW (1981) states that triclopyr "is not considered a persistent compound in soils." However, the available information suggests that triclopyr can be viewed as somewhat persistent. Mullison (1979) states that triclopyr has an average half-life of 46 days in soil, depending on soil type and climatic conditions (no additional data provided). Degradation to trichloropyridinol has a half-life of between 79 and 156 days at 15°C and "less than 50 days" at 25°-35°C (EPA, no publication date provided [e]; Regoli and Laskowski, 1974; Laskowski et al., 1975). Degradation appears to be 5 to 8 times slower than this under anaerobic conditions (Bidlack et al., 1976). After application of 3.36 kg/ha of tricolpyr, Norris et al. (1977) found residues of 350, 172, and 65 ppb afer 6, 9, and 12 months. Degradation of trichloropyridinol to secondary degradation products was shown to have a half-life of 8 to 279 days in a study using 15 soils from ten major agricultural areas (Mullison, 1979).

In the review of registration material, EPA (no publication date provided [e]) states that degradation of triclopyr is primarily by microbial action. As in most situations of microbial degradation, dry soils and saturated soils decrease the decomposition rate, while moist soils increase it (Dow Chemical U.S.A., 1981).

Persistence in Water

Limited data is available on the fate of triclopyr in water. Triclopyr does not readily combine with sediments or other organic materials and will remain in solution once it has entered a body of water (TRW, 1981). Volatilization is insignificant (Dow Chemical U.S.A., 1981). Hamaker (no date) studied the hydrolysis of triclopyr in a buffered aqueous solution at pH levels of 5, 7, and 8, at temperatures of 15°, 25°, and 35°C. He found triclopyr to be stable to hydrolysis for periods of up to 9 months.

This same study found only minor amounts of photodegradation products. This contrasts with two other studies cited by TRW (1981) which state that photodegradation is rapid and complete, with a half-life of 10 hours in water at 25°C (Mullison, 1979;). TRW (1981) states that "photodegradation is a major pathway for the dissipation of triclopyr in aquatic environments."

Indicators of Potential Ground Water Contamination

Table II-50 provides information on parameters associated with the mobility of triclopyr. These parameters, and their associated thresholds, have been suggested by EPA for use in assessing the potential for pesticide contamination of ground water. A discussion of these parameters and thresholds, and the methods for arriving at designated values for individual herbicides, is presented in the main body of the report as part of the discussion of the fate of herbicides in the environment.

TABLE II-50

INDICATORS OF POTENTIAL GROUND WATER CONTAMINATION: TRICLOPYR

Indicator	Value for Triclopyr	Threshold
Solubility	Garlon 3A®: very high; Garlon 4®: emulsifies (Dow Chemical U.S.A.)	>30 ppm
K_{oc}	105.7	<300-500
Speciation at pH 5	Anionic	Anionic (negatively charged)
Hydrolysis half-life	Stable (acid)	>6 months
Photolysis half-life	10 hours	>3 days
Vapor pressure	1.26×10^{-6} mm Hg at 25° C (acid)	$<10^{-2}$ mm Hg

* ND = no data.

4. TOXICITY TO NON-TARGET ORGANISMS

Birds

Dow Chemical U.S.A. (1979, 1981) and Mullison (1979) indicate that triclopyr and its formulations are of low toxicity to mallard ducks and Japanese and bobwhite quail (LC_{50} values for triclopyr, Garlon 3A® and Garlon 4® ranged from 3278 to 11,622 ppm). An 8-day LC_{50} value for the technical triclopyr was reported by Haagsma (1975) to be greater than 5000 ppm for mallard ducklings and 3278 ppm for Japanese quail.

Fish

Triclopyr appears to be non-toxic to fish. Dow Chemical U.S.A. (1979) and Mullison (1979) report 96-hr LC_{50} values for bluegill and rainbow trout exposed to triclopyr and Garlon 3A® ranging from 117 to 891 ppm. Both species were less sensitive to Garlon 3A® than to the active ingredient. A study by Haagsma (1975) cited the 96-hour LC_{50} value for DOWCO 233 to be 148 ppm for bluegill and 117 ppm for rainbow trout. This study also calculated 96-hr LC_{50} values for M-3724 (a water-soluble formulation of DOWCO 233) to be 471 ppm for bluegill and 240 ppm for rainbow trout.

Lower Aquatic Organisms

In studies reported by Dow Chemical U.S.A. (1979) and by Mullison (1979), Garlon 3A® appears to be non-toxic to oysters, with LC_{50} values ranging from 56 to 87 ppm. Garlon 3A® was also non-toxic to shrimp and crabs, with LC_{50} values of 895 ppm and >1000 ppm, respectively.

Soil Microorganisms

A study by Griffith (1976) showed triclopyr to be non-toxic to six soil microorganisms: Aerobacter aerogenes, Salmonella typhosa, Staphyloccus aureus, Pseudomonas aeruginosa, Aspergillus terreus, and Pullularia pullulams. After 72 hours of incubation with 500 ppm, no apparent effect was observed when compared to a control. A field study by Hallborn and Bergman (1979) showed that the rate of nitrogen fixation

of the lichen Peltigera praetextata and its free-living phycobiant algae Nostoc sp. was not significantly affected by treatment with Garlon 3A® at rates typically used in forestry applications.

Potential for Bioaccumulation

Triclopyr and its degradates trichloropyridinol and trichloromethoxypyridine did not accumulate in edible portions of catfish or in fish heads, viscera, or skins in a study by Hedelund (1972). This study also showed that mosquito fish did not accumulate significant concentrations of the residue trichloropyridinol.

Toxicity Data Evaluation

More publicly available information is needed on the carcinogenic, mutagenic, and teratogenic effects of triclopyr before conclusions can be drawn. EPA is currently reviewing its data base on triclopyr and will be identifying data gaps to be filled.

One chronic oral study on triclopyr was done by IBT. EPA considered portions of this study to be valid and decided that the study could be used for supplemental information. Dow Chemical U.S.A. has since replaced the study.

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